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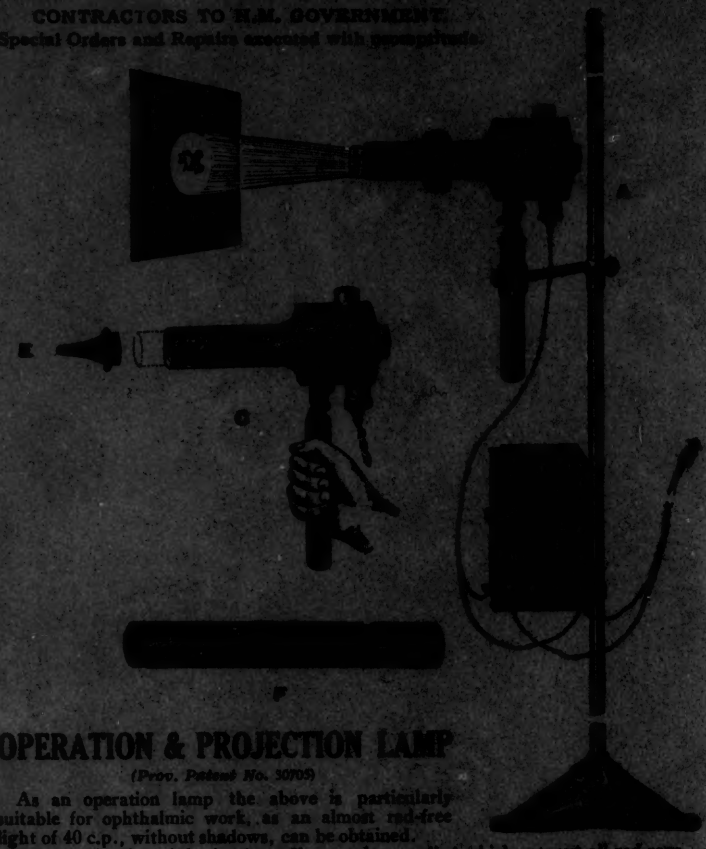

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OCTOBER, 1923

COMMUNICATIONS

THE BLOOD-PRESSURE IN THE EYE AND ITS RELATION TO THE CHAMBER PRESSURE

BY

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THIS paper is a belated sequel to three others which appeared in this journal under the same title in 1917 and 1918. The first dealt with principles governing the pressure of fluids moving through tubes,[1] the second with measurements of blood-pressure in general,[2] the third with the blood-pressure in the retina.[3] The present article re-examines the methods by which the pressures in the central artery and vein have been estimated; it deals particularly with the effects of external pressure on the eye, and with the significance of the "venous pulse."

The use of certain terms. The term " chamber-pressure," occurring so frequently in these pages, stands for " intraocular pressure" : it saves three syllables every time, is not less expressive I think, and avoids any possibility of confusing the pressure in the intraocular blood-vessels with that in the chambers. It signifies, of course, the pressure exerted by the fluid in the aqueous and vitreous chambers against the containing walls—a pressure not measurably different in the two regions.

The condition of the eye which we test with two fingers or the tonometer is universally called the "tension," and the term, though somewhat vague when so used, is far too convenient to be dropped; but is there any good reason for using this word, in otherwise careful writing, as though it were synonymous with "pressure" ? One reads of "intraocular tension," and of the "tension" inside

and outside a blood-vessel. I venture to urge, as I have done before, that such expressions rob the word of its distinctive meaning; they obscure the difference between the pressure of a fluid and the tension of the membrane that contains it.

Properly used the words mean different things: *pressure* implies push, *tension* implies pull. The pressure of the fluid in the chambers keeps the cornea and sclera in a state of tension; the pressure of the blood gives tension to the wall of the vessel. The tension in these cases is due to the pressure, but it is neither identical with it nor always proportional to it. The sclera and the cornea bear equal internal pressure per sq. mm., but the sclera is tenser than the cornea because of its flatter curvature. With equal chamber-pressures a large eye has a tenser wall than a small one. With equal blood-pressure in both, an aneurysmal sac is tenser than the artery that feeds it; if it bursts it does so not only because its wall is weakened, but because it is stretched more forcibly. In short the tension of a membranous envelope is determined not merely by the internal pressure per unit area, but by the size of the cavity and the curvature of the wall in the region in question.

This principle has long been known and stated mathematically (see Leber,[4] Parsons,[5] and for fuller exposition and references Cranston Walker[6]). Objective proof is open to anyone who cares to repeat a simple but striking experiment shown some years ago by Rayner Batten.[7] Slightly modified it is easily made as shown in Fig. 1:—

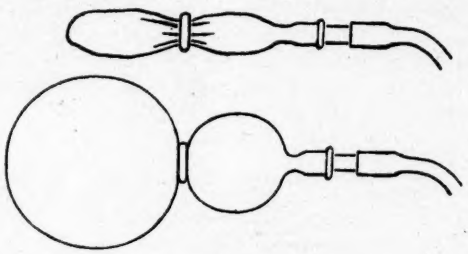

FIG. 1.

Connect a rubber toy balloon with a small hand-bellows. Before inflating it slip over it a small curtain- or finger-ring, placing this rather nearer to one end of the balloon than to the other. Now inflate. The balloon forms two communicating chambers, one larger than the other, and while the larger expands rapidly, its wall becoming thin and tense to the touch of the finger, the smaller enlarges slowly and remains comparatively slack. To prove that this is not due to any initial inequality in the membrane, deflate the balloon and shift the ring to the other side of the middle; then re-inflate. The two parts are now reversed in size; the larger again expands and grows tense—and perhaps bursts—while the smaller remains much less tense. Yet from first to last the pressure per sq. inch is the same in both. Caution:—When the parts differ much in size the larger may drag the smaller through the ring and shoot the ring off with force.

In this experiment the relation of the tension to the pressure alters greatly because the membrane is highly extensible. Under slight inflation the wall of the larger chamber becomes tenser than that of the smaller, and, therefore, suffers more expansion; this increases the inequality of tension and leads to further inequality of expansion, and so on. Fibrous envelopes are comparatively inextensible, but their tension in relation to size and to pressure from within is subject to the same principle.

Blood-pressure in the central artery and vein

Entrance-pressure. The pressure in the central artery has been measured by raising the chamber-pressure artificially, and noting its height when it just suffices to overcome the blood-pressure and close the vessel. Intermittent closure shows that the diastolic pressure is overcome, persistent closure that the systolic pressure is overcome. The behaviour of the artery is watched with the ophthalmoscope.

Clearly this method can be trusted only if the chamber-pressure at the stages mentioned can be correctly measured. In the laboratory it can be raised by known amounts by means of a canula connecting the vitreous with a manometer (von Schultén[8]). In the clinic, the canula being inadmissible, a different method has been employed. The chamber-pressure is raised by pressing on the eye externally with an instrument which, by means of a spring and pointer, indicates the amount of force exerted, and from this indication the chamber-pressure is inferred (Thomson Henderson,[9] P. Bailliart[10]). The clinical method, apart from its manual difficulty, has certain drawbacks. The instrument at best can only measure the *increment* of pressure it induces in the eye, whereas it is the *resulting total, i.e.*, the previous pressure plus the increment, that we want to know; its reading, therefore, must be supplemented by an estimate of the previous pressure. Moreover, the increment is not measured with certainty, for it varies not only with the amount of force exerted, but with the manner of applying it and with the character and condition of the eye.

To what extent do these drawbacks impair its value? The following experiments were undertaken with the aim of answering that question. In considering them the reader will kindly bear in mind that the static force exerted by a spring in any condition of extension or compression can be represented by a weight; also that the pressure of a liquid, though conveniently expressed by the height of a column of water or mercury, is defined more fully in terms of weight and area: a pressure of 20 cm. H_2O means 20 grammes per sq. cm., for 1 cc. of water weighs 1 gramme.

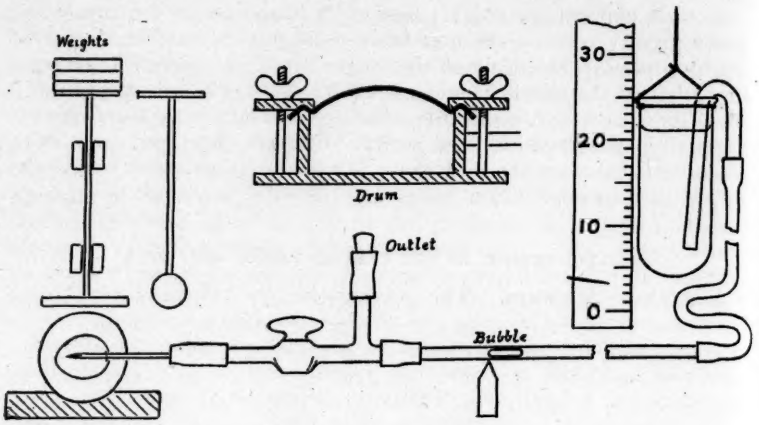

FIG. 2.

Fig 2 shows the nature of the apparatus. The chief tests were made on excised eyes of freshly killed oxen, sheep, and pigs; some on a brass drum covered by a membrane; a few on small rubber balloons.

External pressure was applied by a series of 20-gramme weights placed on a carrier weighing 20 grammes. Two interchangeable carriers were used; one terminated in a flat disc of greater diameter than the area of contact with the eye, the other in a spherical knob 8 mm. in diameter.

The internal pressure was controlled by a water-column, but the readings are here converted for convenience into mm. Hg, the nearest whole number being given; thus the initial pressure of 20 cm. H_2O is given as 15 mm. Hg instead of 14.7. The canula lay in the anterior chamber, where blockage is more easily avoided than in the vitreous, the weight being applied, like the dynamometer in the clinic, over the sclera.

The tubes were freed from air by opening the outlet. The necessary bubble was brought into the horizontal tube by momentarily raising the syphon from the reservoir while the outlet was open. This bubble (von Schultén's device) is essential. To measure the rise of chamber-pressure induced by a given weight one must have the tap open; but an open tap allows fluid to escape through the canula, and this, unless rectified, vitiates the measurement, for there is no such escape in an unpunctured eye. The bubble meets the difficulty. Before the weight is added, the tap being open and the height of the reservoir registered, the position of the bubble is marked. On addition of the weight the bubble moves towards the reservoir, but is immediately driven back to its previous position by raising the reservoir. The eye now contains the same quantity of fluid as before—except for the small amount that has escaped meanwhile through the natural channels—and the height of the reservoir shows how much the chamber-pressure has risen under the added weight.

The results of these experiments may be summarised as follows:—

The rise of chamber-pressure under a given weight varies with the form and extent of the surface bearing on the eye: the smaller the surface the greater the rise. Table I shows the pressures that were found under the disc and knob respectively when they were

equally and progressively weighted. The initial chamber-pressure in every case was 15 mm. Hg. Notice that it always rose more under the knob than under the disc; that the difference increased as the weight increased; and that it was greatest in the smallest eye. In the pig's eye weighted with 80 grammes the rise was 21 mm. Hg greater under the knob than under the disc (average of four eyes tested).

The reason that the disc and the knob raise the chamber-pressure unequally is that they deform the eye unequally. The knob finds a smaller supporting area than the disc, and a smaller supporting area for a given weight implies a higher internal pressure per sq. mm.

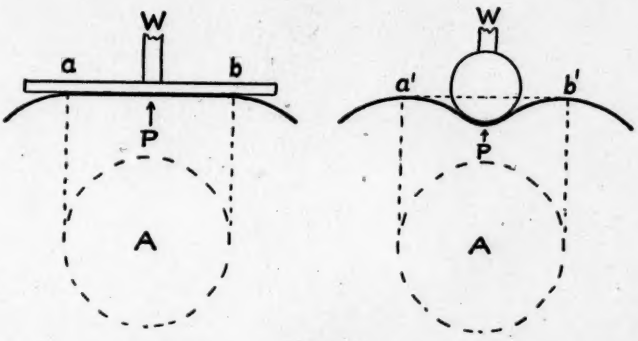

FIG. 3.

Let Fig. 3 represent the disc and knob bearing on two equal membranous spheres filled with water and having no outlet. The disc flattens the membrane down until the flattened part meets with as much pressure from the water below it as from the weight above; then the flattened part supports the weight. The supporting area in this case is the area of contact: (diameter a b). The knob, on the other hand, pits the membrane and sinks into it until the upward pressure against the pitted area equals the weight. Here the supporting area is not the area of contact, but that of the depression as measured on an imaginary plane subtending it (diameter a^1 b^1).

In each case fluid is displaced, and the greater the volume displaced the more is the whole membrane stretched, and the more is the pressure of the fluid raised. The figure shows that when the disc and knob are so weighted as to find equal supporting areas (a b=a^1 b^1) the knob displaces more fluid than the disc: it abolishes not only the space above the line (a b), but also some of that below it; in other words, in order to give equal areas the knob must be weighted more heavily than the disc. It follows that when equally weighted the knob will give a smaller area than the disc, and as already shown, a smaller area for a given weight implies a greater pressure per sq. mm. to uphold it. In each case, according to a well-known principle, the internal pressure per unit area (P) is found by dividing the weight (W) by the number of such units in the supporting area (A), thus $P=\frac{W}{A}$

TABLE I.

Weight in grammes			0	20	40	60	80
Chamber-pressure in mm. Hg.	ox ...	disc ...	15	18	23	28	34
		knob ...	15	20	29	37	43
		difference		2	6	9	9
	sheep	disc ...	15	21	29	36	43
		knob ...	15	25	37	47	55
		difference		4	8	11	12
	pig ...	disc ...	15	22	31	39	47
		knob ...	15	29	44	56	68
		difference		7	13	17	21

A given instrument raises the chamber-pressure less when placed on the eyelid than when placed directly on the eye, and the smaller the terminal the greater the difference. A pig's eye was tested, first bare then covered by two layers of wet chamois leather—a very supple artificial eyelid 1.5 mm. thick. Under the knob the differences were as shown in Table II; under the disc they were smaller still but definite. We tried the same thing with the real eyelid of a pig, but it was so mobile that the knob slipped sideways and the results were worthless. Unquestionably an interposed pad lessens the rise of pressure by enlarging the supporting area, and its effect is proportionally greater as the terminal is smaller, but if the eyelid is at all comparable with the substitute we used, its effect in this way must be small.

TABLE II.

Weight in grammes		0	20	40	60	80
Chamber-pressure in mm. Hg.	eye bare ...	14.7	32.7	47.5	61.0	73.5
	eye covered ...	14.7	31.5	46.0	58.6	69.8
	difference ...		1.2	1.5	2.4	3.7

The rise of chamber-pressure under a given weight varies with the size of the eye: the smaller the eye the greater the rise. This is seen in Table I, if we compare the effects of any given weight on the eye of the ox, the sheep, and the pig respectively. Under the heavier weights the differences are large: under 80 grammes the knob raised the pressure to 43 mm. Hg in the ox's eye, to 55 in the sheep's, and to 68 in the pig's. The reason, in part at least, is easily given: other things being equal, a given weight finds a smaller supporting area on a small eye than on a large one, as shown in Fig 4.

But these eyes differed greatly in size: they were chosen for that reason to demonstrate the principle. Healthy human eyes differ much less. Except in young children and high myopes, the smallest diameter is seldom less than 22 mm., the largest seldom more than 26 mm.,[11]—a ratio of 1 to 1.18, whereas in the case of the pig and ox the average diameters are about as 1 to 1.68. The errors likely to arise through difference of size in human eyes are, therefore, much smaller than the figures in Table I might suggest, but are probably not quite negligible.

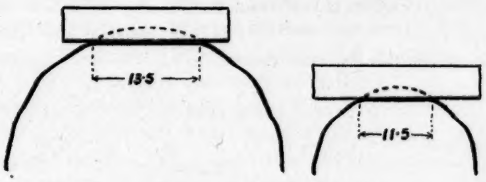

FIG. 4.

Fig. 4 was drawn by measurement from an ox's eye and a pig's eye, each under a flat weight of 80 grammes. An initial chamber-pressure of 25 mm. Hg, was established in each, the tap was closed and the weight applied. The supporting area printed itself on the surface of the weight by means of colour smeared on the sclera. The smaller eye, because of its sharper curvature and lesser expansibility, gave a smaller supporting area than the other, and, therefore, suffered a greater rise of chamber-pressure.

The rise of chamber-pressure under a given weight varies with the character of the eye wall: the less extensible the tissue the greater the rise. An ox's eye was compared with a rubber balloon filled with water, these being about equal in size when their internal pressure was 15 mm. Hg. Under a weight of 100 grammes the chamber-pressure rose to 52 mm. Hg in the eye, and to 23 in the balloon—a rise of 37 against 8 mm. Hg—the wide difference being due, of course, to the high extensibility of the rubber as compared with the fibrous tissue. We also used the drum (see Fig. 1) covered first with sheet-rubber 0.7 mm. thick, and then with the same supplemented externally by very thin silk. The silk limited the extension of the rubber and thereby increased the rise of pressure under a given weight.

But the principle hardly needed demonstration: it is self-evident; the question is whether human eyes differ in this respect enough to affect the findings materially. Schiötz's experiments in tonometry,[12] and later my own,[13] showed that, of eyes having the same chamber-pressure, some are more impressible than others, but they did not show how far this may depend on difference of tissue rather than of size. The only positive evidence on that point relates to the eyes of children. Under continued excess of

chamber-pressure the child's eye, unlike that of the adult, enlarges considerably in all diameters, although by reason of its smaller size its envelope is normally less tense than that of the adult eye (see p. 450); evidently its tissue is more extensible—as one would expect.

Equal increments of weight may induce unequal increments of chamber-pressure in one and the same eye. Having considered the effects of difference in the instruments used, in the manner of using them, and in the eyes examined, we have still to note the effect of a given increment of force at different pressure levels in a given eye. Table III shows the same series of observations as Table I, but gives the *increments* of chamber-pressure instead of the *totals* induced by successive weights of 20 grammes.

TABLE III.

Weight in grammes			0	20	40	60	80
Increments of chamber-pressure in mm. Hg.	ox	disc	3	5	5	6	
		knob	5	9	8	6	
	sheep	disc	6	8	7	7	
		knob	10	12	10	8	
	pig	disc	7	9	8	8	
		knob	14	15	12	12	

In every case the increment was smaller under the first than under the second weight. This was to be expected, for the extensibility of the eye diminishes as the chamber-pressure rises: with subnormal chamber-pressure it is considerable, about the normal level it diminishes rapidly, at high levels it is very small (see measurements in dog, rabbit, and man by von Schultén[8] and Koster[14]). Since the initial pressure was subnormal in our experiments, the first weight would displace fluid more easily than the second, and would, therefore, raise the pressure less.

But under the third and fourth weights the increments, instead of growing larger as one might expect from the lessened extensibility of the eye, fell off again. This can hardly have been due to faulty observation for the tendency was always in the same direction; moreover, it is visible also in Bailliart's charts. Possibly it was due to the altered shape of the loaded eye and a consequent effect on the size of the supporting area; or it may have arisen through increased escape of fluid through the natural channels under increased pressure. Whatever the cause, one sees that under equal additions of force the chamber-pressure may rise by unequal steps.

The foregoing observations show that the clinical method in question cannot be expected to measure the pressure in the central artery with precision. It has, in fact, given some very discrepant

results, but it is not on that account to be hastily condemned, for much of the discrepancy is explained when one compares the instruments employed.

Thomson Henderson's "pressure-gauge"[9] was the first in the field, and through Dr. Henderson's courtesy I have been able to examine the original instrument. Its foot presents a circular flat bearing-surface 3 mm. in diameter. Bailliart's dynamometer,[10] designed later but without knowledge of Henderson's, ends in a button 7 mm. in diameter with a "slightly convex" bearing surface. Applied with equal force to the same eye, these instruments would raise the chamber unequally, Henderson's giving the greater rise because of its smaller foot. Their findings, therefore, are not directly comparable. *A uniform foot* of fully specified and easily checked dimensions for all such instruments (as also for tonometers), would save much uncertainty.

They differ also in their *system of graduation*. The pressure-gauge is marked in mm. Hg, the intention being to show the rise of chamber-pressure induced by the force applied. Such a scale would be invaluable if trustworthy but, as shown above, it cannot be correct for all eyes, and even for the average eye the desired values are difficult to obtain. They can only be found by elaborate trial on many human eyes; no drum or other artificial substitute can give them. The instrument kindly lent me for examination was found, when tested against a balance, to indicate a rise of 3 mm. Hg for each 10 grammes of force exerted—except near to zero. It will be shown below that this value is too small for the human eye and would lead to a considerable underestimate of the effect produced on the chamber-pressure.

Bailliart's dynamometer is marked in grammes and shows the force exerted. This scale has no difficulty for the instrument maker, and can be tested at any time by the user, but it does not show the rise of chamber-pressure. For clinical purposes Bailliart holds this to be unnecessary: he is content to measure the applied force under which the artery closes in the individual case, and to compare it with an ascertained normal average. But he has not left the matter there. With infinite pains he has used a Schiötz tonometer together with his dynamometer on many healthy eyes, in order to learn the rise of chamber-pressure occurring under known increments of force. Taking from his table[10] the trials up to 100 grammes, and allowing for the known loss of force when the dynamometer is used horizontally, one finds an average rise of 6.6 mm. Hg for each 10 grammes of force, and a little may be added because the mercury-values in the Schiötz chart are somewhat too low.

Further, Bailliart (with Magitot[15]) used the dynamometer together with an injection-manometer on a cat under anaesthesia.

The average rise per 10 grammes works out at 8.3 mm. Hg—a result according well with the foregoing, as the cat's eye is smaller than the man's. Certain observations of my own[13] on human eyes after death, with an injection-manometer and an applanation tonometer weighing 10 grammes, are also in agreement; with four different initial chamber-pressures, the average rise under the 10-gramme weight was respectively 7.1, 7.2, 7.3, 7.3 mm. Hg. These findings together with those from the pig, sheep, and ox are placed seriatim in Table IV; they must not be taken as strictly correct, and they are obviously not exactly comparable with each other, but it is significant that the figures found for the human eye place it among the others in accordance with its size and shape. We may be sure, I think, that under an instrument such as Bailliart's the chamber-pressure of normal human eyes rises on the average by something like *7 mm. Hg per 10 grammes of force*, and that under one with smaller bearing-surface it would rise more.

TABLE IV.

Average rise of chamber-bressure per 10 grammes of force applied.

Ox's eye (as described, with flat compressor)			2.4	mm. Hg.
,, ,, ,,	knob	,,	3.5	,,
Sheep's eye ,, ,,	flat	,,	3.5	,,
,, ,, ,,	knob	,,	5.0	,,
Pig's eye ,, ,,	flat	,,	4.0	,,
,, ,, ,,	knob	,,	6.6	,,
Human eye, living, chamber-pressure measured by Schiötz tonometer (from Bailliart's table; *Annales d'Oculistique.*, p. 658, 1917)			6.6	,,
Human eye, post-mortem, chamber-pressures measured by manometer and applanation tonometer (Priestley Smith)			7·3	,,
Cat's eye, living, anaesthesia, chamber-pressures measured by manometer (Bailliart and Magitot)			8.3	,,

So far then, one would expect Bailliart's method to afford valuable though not precise information as to the normal entrance-pressure in the human eye, and in some hands it has, in fact, given nearly uniform results. Bailliart estimates the diastolic pressure in the central artery to be a little over 30 mm. Hg, the systolic a little over 70 mm. Hg; Velter[16] puts them at 35 and 65; Nunes at 30 and 62. On the other hand, Duverger and Barré,[17] apparently following the same method, found higher pressures: diastolic 50 to 60; systolic 80 to 100; no reason for the divergence is apparent. And there is another difficulty.

von Schultén, using a more direct method, found much higher pressures. He raised the chamber-pressure by means of an

injection-manometer, and watched the artery under high magnification (see *Brit. Jl. of Ophthal.*, 1918, p. 258); result: diastolic pressure at least 90, systolic 100 to 120 mm. Hg. True, this was in rabbits' not in human eyes, but mean blood-pressure in mammals is said to differ little and to be independent of size. Whence then the discrepancy? Possibly the following considerations will explain it.

The injection-apparatus makes pressure on the arteries inside the eye, but not on those behind it; the dynamometer makes pressure on both. von Schultén's method raises the pressure in the afferent arteries to some extent, and perhaps considerably, for since the carotid is not far distant from the ocular capillaries, the natural fall between the two must be steep, and the steeper the natural fall the greater the rise above an obstruction (see *Brit. Jl. of Ophthal.*, 1917, p. 658). Bailliart's method includes an additional and conflicting factor: the eyeball, hardening as its chamber-pressure rises, is pressed against the parts behind it. This double influence on the arteries suggests that of two fingers side by side on the radial at the wrist: the force of the stream under the lower finger is at the mercy of the upper. The conditions in the orbit are highly complex; one cannot compute the net result; but it seems possible that the backward pressure of the eye—by slightly bending the arteries at points between the optic foramen and the papilla, as well as by lessening their calibre—may lower their blood-pressure before they reach the eye. In any case the two methods must affect the blood-pressure at the papilla unequally in the act of measuring it.

It is clear that only a rough estimate of the normal pressure in the central artery can be made. The figure, 90 mm. Hg, given as the mean in my earlier paper was based on the findings of von Schultén and Leonard Hill, and was no doubt too high. The following figures are probably nearer to the reality. *Normal entrance-pressure: diastolic, 40 to 50 mm. Hg; systolic, 70 to 90 mm. Hg.*

Exit-pressure. The central vein like the artery is subject to the chamber-pressure and must support it or collapse. In many eyes the so-called "venous pulse" shows that the resistance of the vein near to its exit is alternately greater and less than the chamber-pressure, for each in turn overcomes the other; and since this resistance depends chiefly on the lateral pressure of the blood-stream, we have only to measure the chamber-pressure to learn approximately the exit-pressure of the blood.

But what is the exit-pressure in the many eyes that show no venous pulse? Clearly it is high enough to hold the vein continously open in spite of the oscillating chamber-pressure, and

we have indirect proof that it is usually not much higher. The lightest touch of the finger on the eyelid narrows the vein at its exit (Thomson Henderson[9]), in other words, the wall of the vein yields to a very small rise of chamber-pressure; before the touch, therefore, it must have been only just holding its own.

Further, we know that when the chamber-pressure rises above its normal level the venous pressure rises with it, for the finger even when firmly applied does not close the vein. As the vein narrows, the *vis a tergo* of the blood-stream drives up the venous pressure until it supports the increased load, and the outflow though diminished is not arrested. Only by exerting force enough to close the central artery can the finger permanently close the vein, for so long as there is inflow there must be outflow.

An experiment described in a previous paper illustrates the principle (*Brit. Jl. of Ophthal.*, 1917, p. 661). A piece of pigeon's gut carries a stream of water through a glass chamber filled with water; the pressures in gut and chamber are controlled. A pressure of 15 cm. being established in the gut, that in the chamber is gradually raised from below that level. When it just exceeds 15 cm. it affects the stream through the gut: the distal end of the gut is compressed, the outflow is diminished, the pressure of the stream above the point of compression rises, below that point it falls. *The hindrance begins simultaneously with the narrowing at the exit.*

Again, we have evidence that when the venous pressure is raised by disturbance of the circulation outside the eye, the chamber-pressure rises also. Leonard Hill[18] found (in animals) that a rise of blood-pressure in the vena cava induced an equal rise in the torcular Herophili, and in later experiments he and Thomson Henderson[19] found that the rise in the torcular was associated, after a little delay, with an equal rise in the chambers of the eye.

It appears then that the venous exit-pressure and the chamber-pressure are usually nearly equal, and that when either of them rises or falls the other does the same—a safeguard against oedema and haemorrhage in the eye. Yet they are not quite equal: the varying behaviour of the central vein shows that their relation to each other differs in different eyes, and that it often changes in the same eye. The question is how and to what extent this happens? Please observe that we are not inquiring whether the venous pressure at the papilla can rise much above its normal level—we know that it does so—but whether it can much surpass the chamber-pressure.

There are conditions under which it certainly does so. During free leakage from a corneal wound, the chamber-pressure may sink almost to zero, while the venous exit-pressure cannot fall below that in the orbital veins; there is then an abnormal difference between the two with consequent risk of haemorrhage. In thrombosis of the central vein the venous pressure rises almost to the level of that in the central artery, while the chamber-pressure may not rise at all, and haemorrhage is inevitable; the

glaucoma occurring later results indirectly from the haemorrhage (Inouye, G. Coats[30]). But these conditions are exceptional; we are considering what may happen in normal eyes.

M. Bailliart,[15] after much study of the subject, reaches some surprising conclusions. He places all eyes that show no spontaneous venous pulse in two groups: (1) those in which the exit-pressure is continuously higher than the chamber-pressure; (2) those in which it is continuously lower, and he distinguishes them as follows:—In the former group finger-pressure induces the venous pulse by raising the chamber-pressure to the level of the blood-pressure; in the latter it does not, because the chamber-pressure is already the higher of the two. Further, he holds that excess of exit-pressure over chamber-pressure can be measured in the following way: if the venous pulse is absent, he ascertains by means of the dynamometer how much the chamber-pressure must be raised in order to induce it; if it is spontaneously present, he ascertains the rise that abolishes it. Venous hypertony in the eye is to be detected in this way.

Unless I am mistaken there are weak points here. Such measurements would be instructive if the dynamometer could raise the chamber-pressure to the level of the venous pressure without altering the latter, but if it raises both it cannot determine their previous difference. Undoubtedly the venous pulse, by its coming and going, gives very delicate indications of changes in the mutual relation of these two pressures, but it seems to be at the mercy of influences so numerous and so small that it can hardly, I think, have any diagnostic value. Its causation has been discussed by many writers since it was first observed more than 60 years ago—notably by Donders in the first volume of *Arch. f. Ophthal.*—but not quite conclusively, I think, and to justify the objection raised above I shall venture to reopen the question. In gathering the evidence now available I have been much helped by Bailliart's writings, and by Elliot's recent article in this journal (*Brit. Jl. of Opthal.*, 1921, p. 481). I will first quote certain clinical observations from the latter:—

Of 200 healthy eyes systematically examined in this respect by Elliot, nearly one half showed the "pulse" spontaneously, the slightest rhythmic change of colour being reckoned as such; about half of the others showed it under light finger-pressure; the remainder not at all. When present spontaneously it was usually increased by light finger-pressure, but sometimes abolished. Firmer pressure usually abolished it, but not in every case. Some persons showed it in one eye only; some at times but not at other times. Bailliart found it to occur spontaneously in a smaller percentage of normal persons, Lang and Barrett[21], (Ballantyne also according to a letter lately received from him), in a larger

percentage, but its precise frequency is not important here; the problem is the seemingly capricious conduct of the vein. What are the influences that promote and oppose its pulsatile collapse?

(*a*) *Arterial pulsation inside and outside the eye.* The collapse occurs almost simultaneously with the beat of the carotid in the neck, and, therefore, with the arterial pulse in the eye, the orbit, and the cranium, and it is easy to see that the pulse-wave in these several regions tells on the collapse in different ways.

Inside the eye under normal conditions an arterial pulse is not visible by ordinary methods, but the Gullstrand ophthalmoscope shows it even in the finer branches: the pulse-wave expands them and enlarges their curves (Kuemmell,[22] de Speyr[23]). The Schiötz tonometer shows that with each expansion of the arteries the chamber-pressure rises by about 1 or 2 mm. Hg, and it is under this rise that in many eyes the vein on the papilla suffers more or less collapse. The collapse implies displacement of blood from the collapsing part and the volume displaced must be received elsewhere. It cannot pass upstream, for here the vein is subject to the same momentary increase of external pressure and cannot expand to receive it. It must pass down stream, expanding the vein in the nerve-trunk or quickening the outflow through it, and any influence which opposes this expansion or quickening of the flow outside the eye opposes the collapse on the papilla. *On and round the papilla* branches of the artery often cross the veins and perhaps compress them as they pulsate. Such direct compression, however slight, would tend to check the venous stream above the crossing and lower its pressure below it, so favouring collapse at the exit. I have published examples which seemed to exhibit this effect (*Brit. Jl. of Ophthal.*, 1918, p. 265), but as Elliot could find little evidence of it they were probably exceptional.

In the optic nerve the artery and vein lie for some distance side by side, sometimes in separate connective-tissue sheaths, but usually in a common sheath (Leber[4]), a position in which the vein is likely to be compressed by each pulsatile expansion of the artery, or at least to be prevented from itself expanding at that moment. The arterial pulse in the nerve, in so far as it tells on its companion vein, opposes the venous collapse at the papilla.

In the orbit the veins on their way to the cavernous sinus are subject to pulsatile compression by the neighbouring arteries, the impulse being transmitted by the other contents of the cavity as in the eye, though probably with more loss because the containing space is less completely walled in. Here again the arterial pulse opposes the expulsion from the eye.

In the skull, as in the eye, the arterial wave raises the pressure of the contents and quickens the escape at the outlets; thus, in

the torcular Herophili (in dogs) Leonard Hill[18] found a pulsatile oscillation of the venous stream, and in the internal jugular vein near to its emergence from the skull (in dogs and calves) von Bergmann and Cramer, in 27 out of 31 experiments, found a similar small oscillation (see Helfreich[24]); in each case *the rise of the venous pressure synchronized with the beat of the carotid.* In some degree, therefore, the arterial pulse in the skull supplements that in the orbit in its opposition to the expulsion from the eye.

The influence of arterial pulsation over neighbouring veins is, of course, not peculiar to the region of the eye; it appears to be widely spread. Bailliart[25] quotes Gley as follows: "Les battements des artères exercent une influence sur les veines voisines . . . . on a constaté, en effet, que toute dilatation artérielle donne lieu a une ondulation veineuse." In some organs, indeed, it seems to be essential to a normal capillary blood-flow, for when an artificial circulation is set up it is soon obstructed by oedema unless the driving force be made intermittent in imitation of the heart's action (Leonard Hill). But arterial pulsation is not the only influence that requires consideration here.

(*b*) *Venous pulsation from the right auricle.* The internal jugular vein, just above the level of the clavicle, pulsates under the influence of the right auricle, and tracings taken at this spot show that the pressure is lowest in the vein just when it is highest in the carotid artery (Sir James Mackenzie,[26] p. 102, and Fig. 44). The fall in the jugular synchronizes, therefore, with the collapse at the papilla, and if we imagine it travelling back to the eye we may well suppose, with Bailliart and Elliot, that it takes part in causing the collapse. But can it travel so far? No doubt it has *some* effect at the papilla, and even further back, but a fall that is real and considerable low down in the neck may become merely virtual—a minute lessening of a rise—higher up the stream; and apparently this is what actually happens before the eye is reached. Even at the upper end of the jugular the venous pressure (in animals—see above) already rises a little with each arterial pulse, and in the orbit it is further subjected to the same influence. One can hardly doubt, I think, that the fall due to the auricular diastole is converted into a rise before it reaches the eye, and that at the papilla the venous pressure rises and falls not alternately with, but together with, the arterial pressure.

If this be true, it follows that under the influence of arterial pulsation *the venous pressure and the chamber-pressure rise and fall together*— but not necessarily to the same extent. When the venous pulse is present we may be sure that with each beat of the carotid the pressure in the chambers is rising more than that in the vein; when we see no venous pulse we may reasonably

suppose that their oscillations are more nearly equal, though a different explanation may be the true one—the basic level of the venous pressure and not its pulsatile oscillation may be raised (see p. 466 and Fig. 5).

Imagine on the other hand that these pressures oscillate *against* each other, the one rising as the other falls, and consider what this implies for those cases where, according to M. Bailliart, the venous pulse is absent *because the venous pressure is continuously lower than the chamber-pressure* (see p. 461). At the moment when the chamber-pressure is at its lowest the venous pressure will be at its highest, but still lower than its opponent—they will be nearly equal; at the next moment, the one having risen and the other fallen, they will differ by the sum of this rise and fall, and a trifle more—yet the vein will show no collapse. Under finger-pressure the oscillation of the chamber-pressure will increase [see below, *d* (2)], and the difference will be greater still, and still there will be no collapse!

The wall of the vein could not remain steady under such an oscillation, for when the inside and outside pressures were equal its tension would be nil, and in that condition it would be displaced by a very small change in their relation to each other.

It may be objected that as the venous collapse usually lasts longer than the arterial wave, it cannot depend entirely on the latter. But the sharp "stabbing" pulse of the central artery seen under finger-pressure is abnormal; the normal pulse, if visible, would show a somewhat different character. Moreover, the pointer of the tonometer shows, I think, that the time during which the chamber-pressure is raised is long enough to account for the effect on the vein; one would expect the refilling of the vein to be a little less prompt than the subsidence of the arterial wave.

The influences so far considered are pulsatile, but there are others which are not so. The venous pulse when spontaneously present can usually be abolished by raising the venous blood-pressure, and when absent it can often be induced by raising the chamber-pressure. It is probable, however, that under either of these changes the difference between the two pressures is still quite small.

(*c*) *Disturbance of the circulation.* When the arm is raised, the head lowered, or the neck compressed, or when a forced expiration is made, a venous pulse previously present will often disappear, and at the same time the vein may be seen to swell (for references see Helfreich[24]). Under such circumstances the venous pressure rises and acquires a continuous instead of an intermittent mastery over the chamber-pressure. We know, however, that when the one rises the other rises also, and that when the pressures inside and outside a vein are equal a very small addition to the inside pressure will expand the wall freely; a small excess of the venous pressure over the chamber-pressure, therefore, will account for the swelling of the vein. It is significant, I think, that healthy eyes showing no venous pulse exhibit, as a rule, no such swelling, *i.e.*, no suggestion of even a small excess of venous pressure.

Changes of arterial blood-pressure also affect the behaviour of the vein at the papilla. Thus, raising the aortic pressure (in animals) may induce a venous pulse; ligature of the common carotid has been seen to banish it on the operated side, and to induce or amplify it on the other; nitrite of amyl (in animals and man) will often induce it.[24] These disturbances act no doubt both on the chamber-pressure and on the force of the arterial pulse.

(*d*) *Finger-pressure on the eye* influences the central vein in more ways than one :—

(1) By deforming the eye it raises the chamber-pressure, adding thereby to the burden on the vein and favouring its collapse. But by narrowing the vein at its exit it leads to a rise of the venous blood-pressure and so opposes the collapse (see p. 460).

(2) By raising the chamber-pressure it makes the tunic of the eye tenser, and therefore less extensible under the arterial wave; it thereby augments the pulsatile rise of the chamber-pressure. We have good evidence of this. The pointer of the Schiötz tonometer shows that in normal eyes the average oscillation is about 1.5 mm. Hg; in glaucomatous eyes about 6 mm. Hg (Ballantyne[27]). More conclusive still, it is greater under a heavy tonometer-weight than under a light one in the self same eye. The augmented oscillation of the chamber-pressure favours the collapse of the vein.

(3) By pressing the eyeball against the other contents of the orbit, especially if the pressure be firm, the collapse is opposed, for pressure on the veins behind the eye hinders the outflow through them and pressure on the arteries here probably lessens the force of their pulsation inside the eye (see p. 459).

(4) By continuance it tends to deplete the vessels and the chambers (though exceptionally it causes turgescence of the veins —see Elliot) so that when it is suddenly relaxed, both venous pressure and chamber-pressure drop to an abnormally low level. Thereupon the veins fill up quickly and become over-full, while the chambers refill more slowly, and normal conditions are gradually re-established. The behaviour of the vein, as described by Elliot, is in accord with these changes. In a few cases the venous pulse puts in a brief appearance immediately the finger is withdrawn, showing that the two pressures though lowered are for the moment about equal; but it soon vanishes, for the venous pressure rises more quickly than its opponent. In most cases there is no venous pulse at this moment, for the fall leaves the venous pressure the higher of the two—as may be proved by light re-application of the finger; this raises the chamber-pressure to the level of the venous pressure and the pulse appears.

Figure 5 is an attempt to indicate these varying relations in a diagrammatic way. Let CP (the thicker line) stand for the chamber-pressure, and RV (the thinner line) for the resistance of the vein at the papilla, *i.e.*, its blood-pressure supplemented by its structural rigidity. Whenever CP rises above RV the vein suffers more or less collapse, and the greater the excess of CP over RV the greater the collapse. CP and RV both oscillate, sometimes equally, sometimes unequally, under the influence of arterial pulsation.

PART I shows RV in three different relations to CP, involving respectively a big collapse, a small collapse, and no collapse of the vein. The difference lies, not in the basic level of RV—this is the same in all—but in its oscillation, *i.e.*, on the local effect of the arterial pulse-wave. Imagine the pulsatile expansion of the

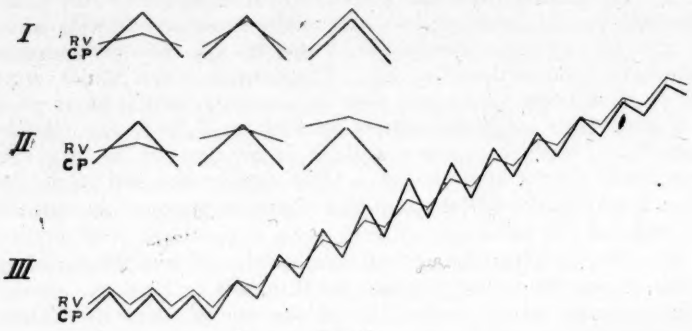

FIG. 5.

arteries to affect the venous stream more powerfully inside than outside the eye, and we have the conditions for a venous pulse at the papilla; imagine it acting equally in the two regions, and we have the conditions for a steady venous stream, for with each beat of the arteries RV will rise equally with CP. Such equality, or something near it, is probably true, I think, of most of the normal eyes that show no spontaneous venous pulse.

PART II shows RV again in three different relations to CP, but here it is the basic level of RV and not its oscillation that differs. Supposing CP to oscillate to the extent of 2 mm. Hg, then a change of 1 mm. Hg in the mutual relation of the two pressures may cause the venous pulse to appear or disappear. Slight disturbances of the circulation will change the relation of RV to CP to this extent, and will explain the varying behaviour of the central vein in the selfsame person.

PART III purports to show what commonly happens when the finger (or the dynamometer) makes gradually increasing pressure

on an eye that shows no spontaneous venous pulse. Until the finger is applied, RV and CP oscillate nearly to the same extent and there is no visible collapse of the vein. Under the finger, they rise together but not quite uniformly; their mutual relation changes, and changes back again; the venous pulse appears, increases, decreases and disappears. The changes are not alike in all cases, for they depend on numerous and varying factors. The diagram stands for the average case and the likeliest explanation of the changes seems to be this:—*Light finger-pressure induces the venous pulse chiefly by augmenting the oscillation of the chamber-pressure; firm pressure banishes it chiefly by compressing the veins behind the eye* (see p. 465 (2) and (3).

This explanation is perhaps incomplete even for the average case. Firm pressure stretches the vessels inside the eye. We know that violent compression of the eye from in front often stretches the membranes sufficiently to rupture the choroid concentrically with the papilla, and it is not unlikely that firm finger-pressure may stretch the central vein enough to add a further hindrance to its blood-flow, and so oppose its collapse. Glaucoma stretches and displaces the vessels at the papilla, but makes no pressure on those behind the eye; its average effect on the venous pulse, if definitely known, would throw light on the question, but the available evidence is insufficient. Bailliart saw the venous pulse in only 7 out of 57 glaucomatous eyes—a much smaller proportion than that for healthy eyes. Fuchs[29] says that it is very often seen; Elliot says that a strong venous pulse is not uncommon, and is often banished by relief of tension. More observations on the point would be helpful.

In almost every eye that shows the venous pulse, one sees proof that a very small factor is enough to turn the scale in its favour or against it. While certain branches of the vein on the papilla pulsate, others do not. These branches can only differ slightly in their structural rigidity, and they can hardly differ at all in their blood-pressure for they are on the point of uniting in a single trunk.

Summary

1. *The arterial entrance-pressure* cannot be measured with precision. The injection-manometer is only applicable in the laboratory; moreover, though it measures correctly, it raises the arterial blood-pressure before it measures it. The dynamometer lacks precision in measuring the chamber-pressure, and perhaps lowers the arterial blood-pressure by compressing the arteries behind the eye. The following estimate is probably near to the truth, but is given with reserve: Diastolic pressure, 40 to 50; systolic, 70 to 90 mm. Hg.

2. *The venous exit-pressure*, under all ordinary circumstances, is nearly in equilibrium with the chamber-pressure. When it falls *below* the chamber-pressure, by more than the small amount which the vein can support by its own rigidity, it is immediately driven up by the *vis a tergo* until the equilibrium is re-established.

When it rises *above* the chamber-pressure, the latter rises also, and equilibrium is approximately re-established, though less promptly and perhaps less completely than in the former case. Any abnormal excess of the venous pressure over the chamber-pressure causes the vein to swell.

3. The self-adjusting equilibrium of pressure inside and outside the vein safeguards the retina against oedema and haemorrhage; when it fails, as during free leakage from the chambers, blockage of the vein, or sudden compression of veins outside the eye, disaster is apt to occur.

4. The veins in the eye are indirectly compressed by each pulsatile expansion of the intraocular arteries; those in the orbit are similarly compressed by the intraorbital arteries. When the effect is equal, or nearly so, inside and outside the eye, there is no visible interruption of the venous stream; when it is greater within than without, blood is expelled from the eye suddenly, and the vein collapses on the papilla—we see the so-called "venous pulse."

5. *The venous pulse,* though essentially dependent on arterial pulsation, is at the mercy of many influences. It is opposed by any condition or change that prevents sudden expulsion at the papilla, and particularly by overfilling or compression of veins outside the eye. It is favoured by all influences that promote such expulsion. The venous pulsation that proceeds from the right auricle tends to favour it, but not in discoverable degree.

6. *External pressure by finger or dynamometer* raises both the chamber-pressure and the venous exit-pressure. It commonly alters their relation to each other, and thereby causes the venous pulse to appear and disappear, but it does not abolish their approximate equilibrium—except when suddenly withdrawn. The amount of artificial pressure which suffices to induce or to banish the venous pulse is not a measure of the difference between the venous pressure and the chamber-pressure.

[Sept. 4.—Since sending this article to press, I have received through the kind courtesy of M. Bailliart, a copy of his new work: "La Circulation Rétinienne a l'Etat Normal et Pathologique" (Paris: Octave Doin, 1923), a volume of 400 pages with a preface by Professor H. Vaquez. In the first part the author summarizes, and in some respects elaborates, his previous studies of the retinal circulation in the normal eye, while in the second and third he discusses its functional disturbances and the various diseases of the retinal vessels. Nothing less than a long and careful notice could do justice to this important work. Here I can only say that my reasons for dissenting from certain of M. Bailliart's conclusions are not affected by it. With much

respect and interest, I submit them as they stand to his consideration, and to that of others who may care to follow up these somewhat subtle questions.—P.S.]

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THE PECTINATE LIGAMENT IN ITS RELATION TO CHRONIC GLAUCOMA

BY

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THE greater part of this report was read at the last Ophthalmological Congress at Oxford, in recognition of Professor Arthur Thomson's well known work on the same subject.

The results are given here of a recent research upon over thirty eyes. The starting point of the investigation was the obtaining of an eye in so early a stage of chronic simple glaucoma that a rare opportunity was afforded of looking for predisposing anatomical causes of the disease, uninfluenced by the secondary effects of high tension. The plus tension began while the

patient, aged 53 years, was in hospital, and was controlled by eserin almost from the beginning, the patient dying a few weeks later.

The most convincing results were got by deep staining with elastic tissue stains. The differentiation thus obtainable seems essential for the demonstration of the finer structure of some of the tissues.

On the whole the best results were got from a particular supply of orcein, discarded years ago because of its incompletely selective action. One of the objections to orcein has been the very variable quality of the supplies obtainable; but by working steadily with the one brand more instructive pictures have been obtained than by any other method. A 2 per cent. or 3 per cent. solution in acid alcohol (HCl 1, methylated spirit 100) has been used chiefly. The solution

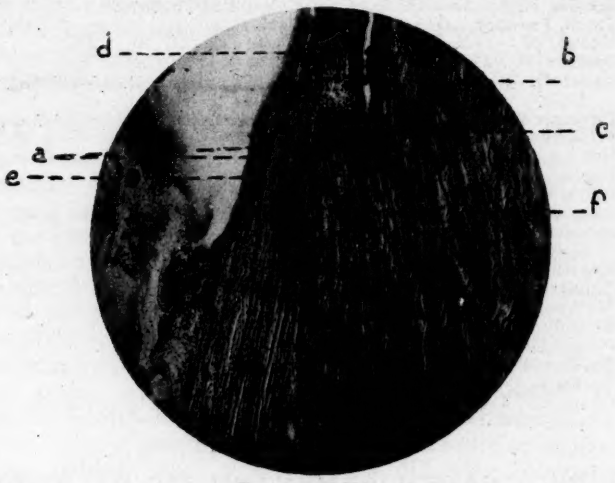

FIG. 1. × 114

containing the celloidin sections was placed in the incubator, or heated slowly over a flame, until of syrupy consistence from evaporation, and then left covered for a couple of hours. The over-stained sections were differentiated in acid alcohol and in alcohol, used alternately till the desired effect was obtained. At one stage each ciliary muscle bundle cut in cross section is seen to be permeated by a close network of elastic tissue, but this mostly disappears if decolourization is carried far enough to show the finer structure of the pectinate ligament.

Fig. 1 is taken from the glaucomatous eye above mentioned. The structure first claiming attention is the rather dark, thick tendon (a). It is clearly the tendon of the longitudinal or meridional portion of the ciliary muscle, which is somewhat unusually well defined in this eye. Imperfect lines of division are seen between bundles alike of muscle and of tendon, separating also the tendon from the adjacent sclera (f). And the longitudinal direction of the fibres of the tendon is well shown by the elastic

tissue stain. Otherwise the sloping line of division between muscle and tendon, though not very sharply defined, might give rise to an erroneous impression of a wide inward-projecting scleral spur or process, forming a complete break in continuity between muscle and pectinate ligament; more particularly since, as in most eyes, the structure of the scleral tissue immediately in contact with the tendon is much more like that of the tendon than is the remainder of the sclera. The two structures are doubtless adherent to one another.

Anteriorly (upwards in the photograph) the fibres of the tendon are seen to pass on without interruption into the slightly looser lamellae of the pectinate ligament. They form a very considerable portion of the width of the ligament.

If the scleral spur or process be defined as the portion of sclerotic forming the posterior boundary of the scleral groove in which Schlemm's canal (b) lies, the angle of the spur (c) is seen to the inner (left) side of the bent end of the canal, separated by a narrow clear line from the tendon. The distinction between sclera and tendon is, therefore, a little more definite at this point.

Quite the innermost trabeculae of the ligament (d), forming a loose network, faint and blurred in the photograph, are seen to be connected by a long narrow elastic band (e), lying on tendon and longitudinal muscle, and bounding the sinus of the anterior chamber with a bundle of muscle fibres seen in cross section. Actually the connection is with a large irregular mass of elastic tissue, very imperfectly stained in this section, forming the main attachment of the oblique (radial) and circular muscle bundles. The elastic band and its ligamentous trabeculae are very distinctly narrower than in most eyes. In this glaucomatous eye, therefore, all the inner more movable portion of the ciliary muscle is placed at a mechanical disadvantage with respect to any inward pull that it may exert upon the lamellae of the pectinate ligament, and, through them, on Schlemm's canal, when the muscle contracts inwards. The mechanical disadvantage consists: (1) in the slenderness of the connecting band and of the trabeculae directly continuous with it; and (2) in the intrusion in the ligament of the broad middle section of lamellae derived from the meridional tendon, separating widely the innermost trabeculae from the outer purely scleral lamellae, which extend forward from the scleral spur; and (3) in the relatively great distance of the muscle, as a whole, behind Schlemm's canal. The angle of the anterior chamber is seen to be placed far back, in relation to the canal.

Sections of the fellow eye, considerably more advanced in chronic glaucoma, present the same features.

The first normal filtration angle, Fig. 2, that became available for comparison under similar staining presents some remarkable contrasts with the above findings. The globe was removed for choroidal sarcoma; tension normal, patient aged 32 years.

The pectinate ligament in this eye is made up entirely of a small inner open, but fairly strong, portion (a) connected with the radial and circular muscle bundles, and a broader outer compact scleral portion (b), into which the angle of the scleral process pushes forward for some distance. There is no definite middle portion of ligament continued forwards from the rather feebly developed meridional muscle bundles (c). The latter have

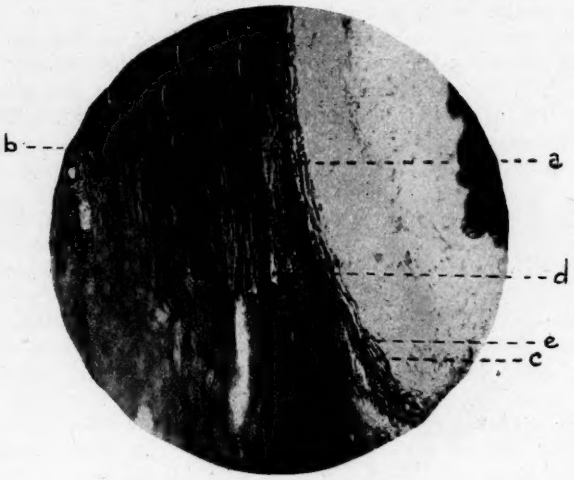

FIG. 2. × 114

no recognizable tendon at all; they undoubtedly extend forward between the sharply defined sclera and the inner loose trabeculae a little beyond the level of the posterior end of Schlemm's canal (d).

The circular muscle fibres in this eye are much better placed for inward traction on the outer scleral portion of the ligament, and, through it, on the canal, even though the angle of the anterior chamber extends rather far back. A strand of oblique muscle fibres (e) can be seen curving forward with the elastic band connecting circular muscle and ligament.

(Schlemm's canal is closed and the scleral lamellae of the ligament are pressed closely together. But this is attributable to the 10 per cent. formalin solution used for fixation of the eye.

The vitreous became swollen, pressing the lens and iris considerably forwards and making the globe tense.)

Fig. 3, from another eye, appears necessary to illustrate extreme inward pull on the pectinate ligament. The canal (a) is widely opened; the trabeculae are separated, and, in front of the canal (above), apparently ruptured. The traction is from cyclitic membrane, a result of old cyclitis, drawing the ciliary processes forward and inward. But the influence on the ligament and canal seem to be directly through the ciliary muscle, and may, therefore, be taken to show fairly well the maximum effect of powerful and continued contraction of the muscle. In some normal eyes the

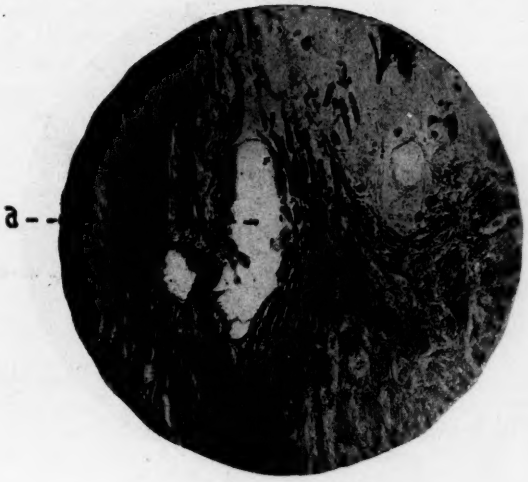

FIG. 3. × 114

muscle is placed almost as far forward as in this eye, the angle of the chamber lying in front of the level of the posterior end of Schlemm's canal.

Fig. 4, from a patient aged 46 years, shows a condition not very unlike the above. (The blurring of the tissues is due to imperfect fixation, the elastic fibres not being cleanly cut. Such blurring, not so readily recognizable under ordinary staining, may have been partly responsible for divergent views upon the anatomy of these parts. Curiously, it appears to be limited always to these particularly highly elastic tissues, and sometimes produces a false appearance of an inwardly projecting scleral process).

The canal (a) is well opened, and all the scleral lamellae of the pectinate ligament (b) are seen to be curved inwards and slightly

separated from one another. Arching outwards to meet these lamellae is a thick band of elastic fibres (c) from the circular and radial muscle. The outer edge of this elastic band almost meets the inner border of the arched scleral trabeculae behind the level of the middle of the canal, to form together nearly the whole of the ligament in front of the junction. Thus, here again the longitudinal muscle, though very well developed, is almost excluded from participation in the formation of the ligament; and in consequence the inner portions of the muscle can exert much more traction on the scleral trabeculae and on the canal than in the glaucomatous eye.

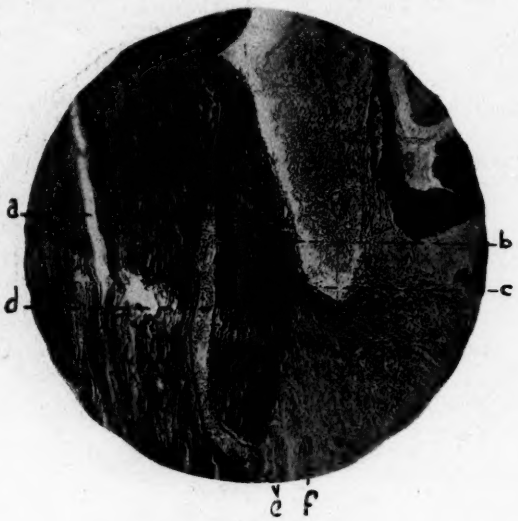

FIG. 4. × 57

The opening of the canal and the arching inwards of the scleral lamellae in this case are due to closely packed blood cells, finding their way out of the anterior chamber after a recent injury. But there is little doubt that a very similar effect could be produced by strong inward contraction of the circular muscle, acting through the well-placed broad elastic band. Inward traction on the ligament is facilitated by the forward position of the muscle.

It may be noticed that in this eye the two outermost meridional muscle bundles (e) are inserted into (or arise from) the sclerotic, rather far back. Another fasciculus (f) runs into the dark patch of fibro-elastic tissue (d) which seems certainly to be a small strip of tendon. The tendency to fibrosis, with advancing years, appears often to be most marked in this position, next to the

sclerotic. The formation may approximate closely to scleral tissue, sometimes containing bands of fibres cut transversely, which may, or may not, be separated from the sclera by meridional fibres continued forwards from muscle fibres. Thus, one approaches to the forward-and-inward projecting scleral spur, which may apparently be sufficiently narrow and prominent--and therefore mobile—to serve in Professor Thomson's well-known "pump action" theory. In accommodation of the eye the process, drawn backwards and inwards by the meridional muscle inserted into it, is supposed to open the ligamentous spaces and Schlemm's canal; and the subsequent recoil of the process by its own elasticity and by that of the attached ligament is expected to force fluid on from the filled canal into efferent veins. There has been no such scleral process among the eyes stained for this research. And without effective differential staining one is very apt to mistake partial tendon-formation for a true scleral spur, so that mistakes are readily made in looking through one's old sections,

FIG. 5a.

especially among those not quite meridionally cut. But from the micro-photographs shown by Professor Thomson at Oxford, there seems to be little doubt that the above description must be accepted as applying to a definite type of eye—one, however, not very common.

The striking anatomical variations here shown and discussed appear to have escaped serious notice hitherto. They possibly include all the main types, but there are many connecting links between the extreme variations.

In the descriptions given in the text above inferences have been suggested. The contrast between the finding in the glaucomatous eye and in the almost normal eyes of Figs. 2 and 4, seems to point in a definite direction. A new "glaucoma theory" is suggested on the assumption that the normal tone of the ciliary muscle keeps up slight continuous traction on the attachments of the muscle. In Fig. 1, represented diagrammatically in Fig. 5a, a predisposition to chronic simple glaucoma may be attributed to imperfect mechanism for drainage, the imperfection being comprised in ineffective action of the circular and radial portion of the ciliary muscle on the pectinate ligament. These inner muscle

bundles, ill-placed and ill-connected, are "crowded out" by the intrusion in the ligament of a broad median strip of lamellae directly continuous with the meridional muscle.

Fig. 5b, corresponding with Figs. 2 and 4, presents the direct contrast to the above. Here the meridional muscle is crowded out from the ligament, and there is nothing to interfere with the traction exerted by the inner more mobile part of the muscle, opening up the inter-lamellar spaces of the ligament and the canal.

The anatomy depicted by Professor Thomson* seems particularly well adapted for drainage. The band from the inner muscle fibres sweeps past the end of the projecting spur and the meridional muscle inserted into the spur and assists in opening up the trabecular spaces and canal. Hence the importance of distinguishing between tendon and scleral process, since in Fig. 1 contraction of the meridional muscle can only have the opposite effect, impeding drainage by drawing the directly connected lamellae tightly together.

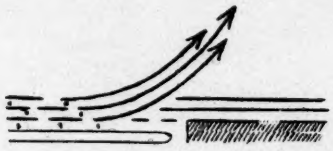

FIG. 5b.

The suggested anatomical predisposition may be regarded as partly individual, but increasing with age as a result of senile fibrosis. Also decline in tone of the muscle with advancing years may be considered to contribute slightly to the glaucomatous tendency. On the other hand, powerful contraction of the circular muscle fibres under the influence of eserin may have sufficient action on the pectinate ligament, even in eyes thus predisposed to glaucoma, to correspond with clinical experience of the influence of the medicament on the tension in chronic glaucoma, an influence varying much in different cases of the malady.

The practical question is: does the suggestion bear investigation? Is there a fairly constant and exclusive association of the anatomy of Figs. 1 and 5a with chronic glaucoma?

Investigation is far from easy. One must expect to find the predisposition in some eyes in which the disease has not yet supervened. On the other hand, some primary glaucomas must occur quite unconnected with this predisposing factor, since this

* See plate facing p. 472 of *The Ophthalmoscope*, Vol. IX (1911), also Elliot's *Treatise*.

anatomical influence is a thing quite apart from the shallowing of the anterior chamber and closure of the filtration angle long established as primary causes of glaucoma. On this account the influence may be considerable in pure chronic simple glaucoma, and quite inconsiderable in acute glaucoma and in the type of the disease which is in the initial stages definitely periodic. That is to say, one may expect it to occur more particularly in the eyes which least often need excision, and least often, therefore, become available for pathological examination. Further, in absolute glaucoma the parts are sometimes so compressed as to afford little information regarding the original condition. And there is sometimes the difficulty, already sufficiently alluded to, of distinguishing between scleral spur and tendon.

In spite of all these sources of confusion, a very definite relationship between primary glaucoma and the particular type of ligamentous connections of Figs. 1 and 5a emerges unmistakably from examination of available old pathological material. Under the circumstances one is a little surprised at the amount of support thus forthcoming for the suggestion. Slides from the (primarily) glaucomatous eyes of over forty patients have been examined in this connection, including a few of the eyes specially stained for this work. More than half the eyes examined belonged more or less definitely to the glaucomatous type, as above defined. That is, a broad section of ligamentous lamellae served as the means of attachment of longitudinal muscle, greatly limiting the direct attachment of the middle and inner muscle bundles; and the muscle as a whole was placed rather far back. Some of the glaucomas to which the description applied were apparently acute from the beginning. On the other hand, in a search among non-glaucomatous eyes this anatomical feature has been but seldom met with.

This paper serves to show how incomplete is our knowledge of the normal anatomy of this most important part of the eye. The influence of age in particular needs precise determination. Uniform fixation and strictly meridional sectioning are essential, if mistakes are to be avoided. On account of insufficient care exercised in these respects, no comment of value can be made upon Thomson Henderson's conception of sclerosis of the pectinate ligament as a cause of glaucoma.

Thanks are due to Mr. Chesterman of Oxford for the excellent micro-photographs shown.

ANNOTATIONS

The General Medical Council and the Teaching of Ophthalmology

The correspondence between the Council of British Ophthalmologists and the General Medical Council concerning the teaching of ophthalmology, published in the September number of this journal, emphasizes the difference of opinion held by these two bodies. The question has been under discussion in our columns on several occasions during the past four years, but it is of such importance to the medical profession and to the public that we feel more than justified in directing attention to it anew.

In 1919, after an exhaustive inquiry into the regulations governing the teaching of ophthalmology and the examinations of students therein in the medical schools of this country and abroad, the Council of British Ophthalmologists submitted to the General Medical Council the recommendation that: " no student shall be considered to have passed the qualifying examination unless he has shown a sound knowledge of practical ophthalmology in an examination conducted by ophthalmic surgeons." (*vide Brit. Jl. of Ophthal.*, vol. 3, p. 165, 1919). It is gratifying to note that this opinion has been emphatically endorsed by the Departmental Committee on the causes and prevention of blindness in their report, published last autumn (*Brit. Jl. of Ophthal.*, 1923, p. 93). That body was impressed by the evidence brought before it "of the inadequate knowledge of diseases of the eye possessed by the large majority of the medical profession on admission to the register . . ." The Council of British Ophthalmologists deemed the appearance of this report a fitting occasion on which to bring the question once more before the General Medical Council; hence the correspondence referred to above.

The letter from the Registrar of the General Medical Council, which we assume emanates from the Council, is not encouraging. The recommendations of the Departmental Committee have apparently been shelved by the well-worn formula "it is too soon to judge of the effect of the resolution recently adopted by the Council." Moreover, the last sentence of his letter, which seems to suggest that credit is due to the Council for anticipating the wishes of the Committee, is distinctly misleading. The Departmental Committee's "wishes" ("recommendations," in the Report) were not that "each student should receive instruction in the diseases of the eye, refraction, and the use of the ophthalmoscope," but that "every student presenting himself for a qualifying examination in medicine shall be examined in ophthalmology."

We confess that we are unable to grasp the mentality of the General Medical Council in reference to compulsory examination in ophthalmology. The experience of many examining bodies has shown not only that it is desirable, but that it can be conducted without difficulty. We venture to hope that, failing action by the General Medical Council, universities and diploma-granting corporations may recognize the desirability, or indeed the necessity, of instituting such examinations as an integral part of their qualifying examination.

Ophthalmic Research in the Combatant Services

A valuable and interesting discussion on ophthalmology in its relation to the Navy, Army, and Air Force took place at the British Medical Association meeting at Portsmouth. We have already (*Brit. Jl. of Ophthal.*, p. 448) mentioned the names of the chief speakers on behalf of the various services and in the ensuing discussion. The President of the Section, Sir John Parsons, pointed out that during the war innumerable ophthalmic problems arose in connection with all three services. In most cases the correct solution could only have been attained by prolonged research, which at that time was often impossible. Action had to be taken on data which were incomplete and faulty. In many cases, notably in the Air Force, ineffective action was attended by serious loss of life and the destruction of valuable material. So impressed were the authorities—and the same applied to the American and other governments—with the imperative need for research in these matters that exhaustive investigations were initiated and are still being carried on. The importance of similar problems arising in connection with naval and military affairs does not impress the authorities with the same dramatic intensity. Yet it is obvious that they should be attacked, and that the proper time to attack them is in peace time. Even in these exigent times money is forthcoming for Industrial and Medical Research, and experience shows that it could not be better invested. The efficiency of the combatant services is a supreme duty which no statesman should dare to neglect, but it can only be attained by constant watchfulness and enterprise, and these are liable to sink into apathy and inertia in the comparative security of peace. The meeting signified its appreciation of the validity of these arguments—so ably exemplified in the remarks of Air-Commodore Munro, Wing-Commander Clements, Gen. Sir Wm. Macpherson, Major Gurley, and Surgeon-Commander Breton—that a resolution urging the Government to carry on research in these ocular problems was carried unanimously.

ABSTRACTS

I.—METASTATIC GONORRHOEAL IRITIS

(1) **v. Hippel, E. (Göttingen).—On a case of unusually severe gonorrhoeal iridocyclitis and optic neuritis. (Über einen Fall von ungewöhnlich schwere gonorrhoischer Iridocyclitis und Neuritis optica).** *Arch. f. Ophthal,,* Vol. XCIV, part iv. 1917.

(1) **v. Hippel's** patient was an athletic man of 18 years who developed rheumatism in the right ankle joint four weeks from the beginning of an acute attack of gonorrhoea. Four days later on waking he found he was almost completely blind. He was then found to have much swelling and injection of the bulbar conjunctiva with some haemorrhages. The palpebral conjunctiva was not so much affected and there was a scanty purulent secretion, in which no gonococci were found. Diagnosis, metastatic gonorrhoeal conjunctivitis. In addition there was much exudate in the pupillary area and synechiae had already formed. The right ankle and knee joints were swollen and painful and the temperature was high. Under treatment the pupillary exudation rapidly cleared and the pupils dilated to the maximum, but the anterior chamber and vitreous were full of opacity. The iritis frequently relapsed, and it was not until two months later that the condition of the vitreous allowed an ophthalmoscopic examination. This showed a high degree of papillitis. Vision was only finger counting, and a large relative central scotoma was present. The temperature remained raised for two and a half months from the onset of the disease. The general treatment consisted in urethral irrigation and the injection of arthigon in increasing doses. Each of the injections was followed by a rise of temperature. A month later a white exudation was found near the macula. This suggested the possibility of a syphilitic infection, but the Wassermann test was negative. Sweating and mercurial inunction were carried out for several weeks. No improvement in the eye resulted. Ten months later and two months after the cessation of atropin the iris was very atrophic and remained widely dilated. Two and a half years later the iris was very atrophic, the media hazy with definite cortical opacities in the right lens. The papillae were atrophic with ill-defined borders. Vision R.E. 2/50 L.E. 2/10.

v. Hippel points out that the rarity of the case lies in the early onset of the ocular symptoms, and the lasting nature of the results of the optic neuritis. He is inclined to doubt the value of specific treatment by arthigon and vaccines, and even suggests that the

fever reaction produced may indicate that the treatment is actually harmful in such cases. No gonococci were found in cultures taken from the patient's blood.

E. E. H.

(2) **Cobbledick, A. S. (London).—Chronic gonorrhoeal infection of the prostate: notes on its persistence and on its relation to recurrent iridocyclitis.** *Lancet*, March 2, 1918.

(2) It was shown by **Cobbledick** in an investigation carried out in 1912-13 (*Ophthalmoscope*, Dec., 1912 and June, 1913), that in a series of nine cases of rheumatic iridocyclitis the gonococcus was present in every one, after periods varying from 7 to 30 years from the date of the last infection. "This investigation," says Cobbledick, "brought to notice a class of gonorrhoeal infection to which little attention has been given. The reason is no doubt due to the fact that they only come under the notice of the ophthalmic surgeon, who in treating them has only treated a symptom, leaving the aetiological factor undiscovered and untreated."

Cobbledick relates some further cases, not all of which are cases of irido-cyclitis, in which the latency of the gonococcus seems to be proved up to the hilt. Longevity of the gonococcus is, of course, no new thing, but from Cobbledick's investigations he concludes that it is capable of residing alive in the prostate without any time limit at all. There may be no perceptible urethral discharge and yet the prostate and vesiculae may be the seat for many years of a gonococcal infection with all other parts of the urinary system unaffected. In investigating cases of iridocyclitis of obscure origin it is the duty of the ophthalmic surgeon to obtain the expert assistance of the genito-urinary surgeon. Massage of the prostate per rectum must be performed, a procedure which, as the author drily remarks, is not popular, and the urine which is passed thereafter must be centrifugalized, and the deposit examined by an expert bacteriologist for the presence of the gonococcus.

Cobbledick's work is of such a nature that nothing would be gained by abstracting the case histories. These must be read in detail to appreciate the conviction which they carry to the mind a conviction, in the author's own words: "that in these so-called rheumatic cases a systematic examination would always reveal, quite apart from the presence of a gleet, a prostate infected by the gonococcus, provided the technique suggested is followed, and I do not doubt that the eradication of the gonococcus would in all probability prevent the recurrences which are such a feature of the disease."

ERNEST THOMSON.

II.—DEVELOPMENT OF IRIS

(1) **Streiff (Jenna).—On a surface depression in the lower part of the iris and on types of iris and transition forms to abnormalities. (Ueber eine untere Irismulde und über Iristypen und Uebergänge zur Anomalien).** *Klin. Monatsbl. f. Augenheilk.*, Vol. LIV, p. 33, 1915.

(1) The depression referred to is regarded as developmental in origin, and has been found by the author always in the typical position—downwards or slightly inwards and downwards. It is simply a partial congenital coloboma of the iris. In connection with iris pigmentation three types are distinguished: *Heterochromia Simplex* in which both eyes are normal or it is doubtful which is the abnormal one; *Heterohyperchromia* in which the darker eye shows a wart-like formation of the iris surface or melanosis sclerae is present indicating—at least for Germans—that the abnormality is in this eye; and *Heterohypochromia* which appears to be the familiar form of pale depigmented iris associated with chronic cyclitis and cataract. This last form the author regards as the result of a pathological course of the pigmentation process dependent on congenital abnormal conditions, as distinct from albinism, which is a cessation of the normal pigmentation process. Under structural types and anomalies of the iris the various modes of arrangement of the trabeculae are discussed. Two chief types may be recognized, the *radial* in which the trabeculae extend to the pupillary margin and the *frilled* or *bordered* form (*Krausentypus*), in which a frill or rim occurs at a little distance from the pupil. This is due to the rarefication of the superficial layer over the circumpupillary zone and may be hereditary. The author has found the frilled type more often in myopes, and the radial type more commonly in hypermetropes. Cases of glaucoma simplex have usually the radial type. Finally, the author discusses the developmental relations of these variations in the iris structure. The paper is illustrated by several drawings, and an excellent coloured plate.

H. M. TRAQUAIR.

(2) **Cirincione, Prof. Speciale (Cagliari).—On the development of the muscles and of the posterior layers of the iris. (Sullo sviluppo dei muscoli e degli strati posteriori dell'iride.)** *Annal. di Ottal. e Clin. Ocul.*, Jan.-Feb., 1922.

(2) Cirincione in this important and beautifully illustrated paper begins with a review of the literature of the subject. As Fuchs pointed out in 1885, even as early as that period, there was no part of the human body of similar size on which so much had

been written; as Cirincione now points out the output of literature on the same portion still flows freely. Cirincione in his capacity of Director of the Ophthalmic Clinic in the Royal University of Cagliari, has access to an extensive embryological collection of material, and has availed himself of this to make exhaustive histological examination of the iris at all stages of development. He sums up the results of his work as follows:—

"1. The first indication of the iris is to be found at the beginning of the fourth month of intrauterine life, and is formed from the marginal tract of the secondary optic vesicle, separated from the posterior investment of the cornea in consequence of the formation of a delicate intermediary fissure. Scanty mesoblastic tissue and a vessel with very delicate walls remain attached to the small tracts of separated epithelial layers. The fissure, however, does not extend beyond the level of the vesicle, and when studied *in toto* (reconstructed) has the appearance of an annular fissure (primordial anterior chamber).

2. The two epithelial folds at the edge of this primordial iris present, at the point where the one fold joins the other, a tract more delicate and formed from a single layer of cubical elements becoming successively less pigmented. It limits a circular space (annular sinus of Szily).

In the foetus of 90-95 mm. the elements of this passage tract proliferate rapidly in the neighbourhood of this edge and are transformed into a germinative zone, to which is due the prolongation of the optic vesicle and the formation of the sphincter muscle.

3. The sphincter of the iris is represented, during the fourth month, by an epithelial slab in the form of a ring, in which pigmented elements, elongated and parallel to each other take origin from the germinative zone and stand in direct continuation with it. During the fifth month the elements of this slab become still more elongated, and are transformed into fibro-cellules inclined with respect to the surface of the iris. In the sixth month the rarefaction of the pigment elements of the slab, and the penetration of connective tissue elements between the fibres of the sphincter determine the process of splitting it up into muscle bundles. All connection with the epithelial layer is only lost in the last two months of intrauterine life.

4. The epithelial layers of the posterior surface of the iris are normally two in number and both are pigmented. These develop differently and separate in the adult structure.

5. The posterior epithelial layer is formed from the proliferation of the elements of the germinative zone nearest to its edge which are generally bent back as the edge advances in development. It is, therefore, a mistake to describe that layer as the

pars iridica of the retina, since the internal layer of the optic vesicle, from which the retina is derived, stops at the ciliary body and is never pigmented.

6. The anterior epithelial layer of the iris is formed from two tracts: one underlying the sphincter, formed in the same way from the proliferation of the germinative zone, the second, on the other hand, derived from the transformation of the voluminous elements of the pigmented layer after it has given place to the pre-epithelial membrane; thus the different aspects that that layer presents in the adult are to be correlated with the differences in development of the pre-epithelial membrane.

7. The pre-epithelial membrane (dilatator according to some authors) is always derived from the underlying epithelium, but does not present a constant structure in the adult. This fact is due to the varying degree of the reunion of the cells of the anterior epithelial layer from which it derives its origin.

The differentiation is most frequently limited to the basal portion of the cell which portion is fused with that of the neighbouring cell so as to give origin to a continuous membrane, refractile, homogeneous, and placed on the back of the remaining cellular elements. At other times the cellular portions are transformed into concealed and arcuate protrusions, which covering each other like roof tiles (*embricandosi*), do not form a true membrane, but form instead in front of the epithelial cells a protoplasmic layer which is obliquely striated, and in which the limits of the single portions which constitute it may be recognized. A final variation occurs in which all the epithelial cells are transformed into an element so crushed that it is adherent obliquely to the back of the neighbouring elements and in sections gives the appearance of a fibro-cellule.

Each of these three appearances may be met with alone in a perpendicular section of the iris, and this is the most common occurrence, but, on the other hand, less frequently all three may be present simultaneously in the same section. This is the explanation of the tenacity with which various authors have denied the appearance of one or the other structure of the pre-epithelial membrane of the iris.

8. The dilatator of various authors and the muscular sphincter, in spite of both having an epithelial origin, differ histologically, inasmuch as while the pre-epithelial elements which are transformed into fibro-cellules and give origin to a true muscular structure, are entirely absorbed in the formation of the sphincter; only a portion of the epithelial elements, on the other hand, (exceptionally the internal cell, which does not in every case retain its proper pigment) goes to the formation of the so-called dilatator, and the membrane thus formed, lacking nuclei, must be

regarded as a contractile epithelial membrane with a muscular function."

A useful bibliography, going back to Bruch in 1844, accompanies the paper as well as five coloured plates. E. E. H.

III.—ENCEPHALITIS LETHARGICA

(1) **Zarzycki, Pierre.—Ocular complications of cerebrospinal meningitis. (Des complications oculaires de la méningite cérébrospinale.)** *Arch. d'Ophtal.*, July-August, 1918.

(1) **Zarzycki** reports five cases observed by him in a general hospital since the beginning of the war, giving clinical notes of each. The ocular manifestations were: (1) iridoplegia incomplete, with bilateral papillitis; (2) neuro-retinitis of unequal severity in the two eyes; (3) intermittent strabismus, with glaucoma, followed by shrinking of the eyeball; (4) suppurative dacryo-cystitis; (5) bilateral metastatic ophthalmitis. J. B. LAWFORD.

(2) **Terrien, F.—Purulent irido-choroiditis and meningococcaemia. (Irido-choroidite suppurative et méningococcémie.)** *Arch. d'Ophtal.*, July-August, 1918.

(2) **Terrien** published a detailed clinical account of this case, with a report of the microscopic examination of the affected eyeball.

The case presents several points of interest. It occurred in a healthy male aged 18 years. The ocular lesions showed themselves on the third day and rapidly became severe: the iris was swollen and adherent, a large hypopyon formed, vision was reduced to hand movement. Although the cerebro-spinal fluid showed no abnormality and remained sterile, haemoculture revealed a characteristic growth of meningococcus. Treatment by anti-meningococcic serum injected into the spinal canal and subcutaneously was followed by amelioration of the general symptoms. No improvement in the ocular conditions occurred until after the injection of 4 mm. of serum into the A.C. This was followed by disappearance of the hypopyon and of the exudation in the pupil.

J. B. LAWFORD.

(3) **Dickinson, Dr. G. (Syracuse, N.Y.)—Ocular notes on lethargic encephalitis, with two case reports.** *Amer. Jl. of Ophth.*, p. 587, August, 1920.

(3) The difficulty of differentiating between lethargic encephalitis and syphilis of the central nervous system is of special interest to the ophthalmologist, and **Dickinson** gives a report

of two cases which presented trouble in diagnosis. The first case was that of an apparently healthy man of 26 years who was referred to the author on account of sudden diplopia. No history of venereal or other disease. Pupils were active to light and accommodation; fundus normal, and refraction practically normal. Limitation of external movement of the right eye, and marked esophoria were present. With rest and attention to diet, esophoria almost disappeared, and diplopia ceased, in about a week. Recurrent attacks of somewhat longer duration occurred, the paralysis shifting from one muscle to another, and patient was ultimately referred to a neurologist who diagnosed lethargic encephalitis. Wassermann reaction was negative. Under treatment by the neurologist, muscle balance became normal, and the patient remained well. A slight drowsiness passing into some confusion of ideas during the early part of his treatment by the oculist were put down to atropin idiosyncrasy, but in the light of the later diagnosis these symptoms were attributed to the underlying pathological condition.

The second case was that of an unmarried woman of 33 years, who was healthy, but who had led a sedentary life. The first symptom noticed was that her left pupil was larger than the right. On examination the left eye showed a typical Argyll Robertson pupil. Vision normal; external muscle balance normal, and fundus showed nothing unusual. Field of vision normal. Blood tests negative for syphilis. Beyond a general slowness in reaction time, and a tendency to drowsiness nothing definite could be determined, but the diagnosis of a mild case of lethargic encephalitis was returned by the neurologist. After a course of treatment extending over seven weeks the condition had improved, and the pupil reacted to light almost completely.

Dickinson thinks it would be interesting to know how many mild cases of this disease have been diagnosed as syphilis, and treated as such, and *vice versa*. The fact of the predominating symptom of both conditions being partial or complete paralysis of the external ocular muscles (particularly those supplied by the third nerve), confuses the diagnosis. The fact, however, that in syphilis the paralysis is of an intractable nature forms a guide, the paralysis in encephalitis yielding more readily to treatment. The typical Argyll Robertson pupil met with in the second case refutes the doctrine that this symptom is invariably a luetic one.

J. HAMILTON MCILROY.

(4) **Symonds, C. P. (London).—Optic neuritis in encephalitis lethargica.** *Lancet*, December 18, 1920.

(4) **Symonds** first of all refers to the fact that he has found very little reference in the literature of this disease to changes

in the fundus oculi or disturbance of visual function. Macnalty (L.G.B. Report for 1918) failed to find one case of true papilloedema in 50 cases of the disease in which the fundi were systematically examined. Nor have Morax and Bollack found impairment of acuity or intraocular changes. Buzzard has reported one case of papilloedema in proved encephalitis lethargica and one in an unproved case. Vincent had two cases of grossly impaired acuity but without fundus examination. Bramwell has not had experience of actual optic neuritis in any proved case of this disease.

Symonds then records very fully four cases, seen in America, in which "definite changes were seen in the fundus oculi, together with clinical signs and changes in the cerebro-spinal fluid which favoured their inclusion in the encephalitis lethargica group." The remainder of the paper is taken up with arguments in proof of the correctness of the diagnosis of encephalitis lethargica in these four cases, and the author summarizes his observations, and concludes that his cases are consistent with the diagnosis of encephalitis lethargica if the definition of Barker, Cross, and Irwin be accepted. This definition discards the title encephalitis lethargica and includes encephalitis, encephalomyelitis, polyneuritis, and meningo-encephalo-myelo-neuritis. These authors assume as the cause a single infectious agent which attacks the central and peripheral cerebro-spinal nervous system and its coverings in a widespread, though at the same time a patchy or disseminated way.

In an editorial note in the same issue of the *Lancet* reference is made to Symonds' article, pointing out that Symonds depends for his diagnosis chiefly on the exclusion of other possible diagnoses, but that other means are in sight now that experimental proof is complete that the disease is caused by a living virus. McIntosh (*Brit. Jl. of Exper. Path.* Oct., 1920) has described the successful inoculation of one monkey with material from another, which had been inoculated from a human case and had developed the disease. It remains to discover the organism.

ERNEST THOMSON.

IV.—OCULAR PROTHESES

(1) **Valois.—Oculo-palpebral prothesis; a mobile artificial eye. (Prothèse oculo-palpébrale avec oeil artificiel mobile).** *Arch. d'Ophtal.*, May-June, 1917.

(1) This is a description of an ingenious apparatus, devised by **Dr. Valois,** which was made and fitted to a soldier in whom, as the result of severe burns by molten metal, the right eye was

destroyed and total symblepharon was produced. When cicatrization was complete, the eyelids were adherent in their whole extent, but beneath them the stump of sclera, after evisceration of the contents of the globe, preserved its movements and these movements were transmitted to the eyelids. A very accurate mould of the whole orbital region was made, and it was possible to determine that the maximum movement was limited to a small central area. An artificial eye was constructed of hemispherical shape, the posterior surface of which was in very accurate coaptation with the skin of the adherent lids, so that the movements of the latter were transmitted to the eye. The eye was kept in position by light pressure from an additional and surrounding portion of the apparatus which represented the normal lids and palpebral fissure, and which was placed over the eye. The whole mechanism was attached to a spectacle frame, by which it was maintained in position.

The description of the apparatus is rendered easy of comprehension by two diagrams. The cosmetic result is said to have been satisfactory. It is regrettable that a photograph of the patient wearing the spectacles has not been added.

J. B. LAWFORD.

(2) **Ramsay, A. Maitland (Glasgow).—An oculo-palpebral prothesis.** *Lancet*, May 12, 1917.

(2) **Ramsay**, after referring to the different methods of forming an oculo-palpebral prothesis, and especially to the case reported by Sir W. Collins (*Lancet* 1916, 1, p. 1217), and to the article by Coulomb and Ruppe (*Annal. d'Ocul.*, April, 1916), says that for various reasons he has adopted vulcanite. The parts to be reproduced are carefully modelled in wax, and from the model a plaster of Paris impression is taken. The soft rubber is then moulded on the plaster of Paris and thoroughly vulcanized. The artificial eye is cemented to the vulcanite lids, and after these have been coloured to match the surrounding parts, the prothesis is attached to a spectacle frame. The lens in front of the sound eye corrects any error of refraction which may be present, while that in front of the artificial eye is of the curvature which is found to give the best cosmetic result. Four cases, all of them soldiers are described and illustrated with photographs.

ERNEST THOMSON.

(3) **Roche, Charles.—Improved ocular prothesis. (A propos de l'amelioration de la prothèse.)** *Arch. d'Ophtal.*, Jan.-Feb., 1918.

(3) In this article, accompanied by a number of photographs illustrating the results obtained, **Roche** describes in detail the

method of operating which he employs. He advocates the implantation of a graft of fat taken from the buttock (the fat graft of Sourdille). He operates under local anaesthesia induced by the deep injection in the orbit of a 0.5 per cent. solution of cocain, and by a subcutaneous injection of the same strength in the buttock. He begins by the excision of the graft which is wrapped in a sterile compress. He then removes the eyeball in the ordinary way, inserting a purse string suture. The graft is then placed in position and the suture tightened and tied. Not infrequently two additional stitches are inserted, in the conjunctival wound. The author is well satisfied with the immediate results he has obtained, but expresses some doubt as to their permanence.

J. B. LAWFORD.

(4) **Poulard and Real.—Ocular restoration. Enlargement and adaptation of the socket for reception of the artificial eye.** *Annal. d'Ocul.*, Vol. CLV, January, 1918.

(4) In the present article **Poulard and Real** express their disappointment with the results generally obtained by the use of epidermic and dermic grafts, and of movable glass or rubber masses placed in contracted sockets, and bring forward a piece of apparatus which they have devised for the purpose of keeping the cicatricial bands, once cut, from re-forming, and through the medium of gradually larger casts, of actually enlarging the socket in any required direction.

A metal plate, held in place by head bands (one of which has a spiral spring and goes under the occiput), is carefully moulded over the eyebrow and forehead on the affected side; to this plate is fused a metal sleeve in which runs vertically a steel rod controlled by a screw; near the lower end of this rod is an antero-posterior sleeve in which runs sagittally another steel rod also controlled by a screw—the posterior end of this fore and aft rod carries a metal disc on to which is fastened the plastic material, and later the vulcanized cast of the socket. By means of these two rods, sleeves and screws, any height or depth in the socket can be obtained. A preliminary mould in three pieces of the socket is obtained, then the bands are cut across and a mass of softish material on the disc is pressed into the socket to distend it completely, especially at the parts where the bands are. The cast of the socket thus obtained is in a few days replaced by a vulcanized one, which is worn night and day for ten days without discomfort, any adjustment to the rods being made as needed, and the plastic mould having been altered in any way to give the correct effect. The usual course lasts fifteen days, supplemented by nocturnal wearing of the splint and obturator for some months afterwards to keep the effect obtained. If the socket has no bands

so much the easier will the case be, while narrow palpebral fissures can be comfortably enlarged by the use of gradually increasing sizes of casts.

The article is well illustrated, but the name of the maker is not stated, nor are any details of cases given.

W. C. SOUTER.

BOOK NOTICES

Privy Council. Medical Research Council. Second Report of the Miners' Nystagmus Committee. London: His Majesty's Stationery Office. Price 9d. net. 1923.

The Miners' Nystagmus Committee, appointed in 1920 by the Medical Research Council, issued a first report in 1922, which was fully reviewed in these columns in June of that year. The report in question dealt generally with miners' nystagmus as an occupational disease, and concluded that defective illumination was the essential cause of the disease, and that improvement in lighting was the preventive.

The second report now issued by the Committee deals with two subjects: (1) Incapacity from miners' nystagmus; and (2) relative importance of errors of refraction in miners' nystagmus. Both sections appear under the name of Mr. G. H. Pooley, a member of the Committee, but the introduction states that another member of the Committee, Dr. T. L. Llewellyn, had helped with part one, and that the Committee is in entire agreement with it.

1. As regards the incapacity from miners' nystagmus, a distinction is drawn between the prevalence of physical signs distinctive of nystagmus, and the existence of incapacity arising therefrom. Information as to the former can only be obtained by the medical examination of underground workers. Romiée in 1877-1908 in four Belgian pits found a high percentage of coal-getters suffering from nystagmus. When he examined the coal-getters in the same pits in 1908, after the lamps had been improved in three of them, the proportion affected had fallen considerably, but had not fallen in the fourth pit where the lamps had not been improved. The figures are interesting and may be quoted in full:

Percentage of coal-getters showing nystagmus in four Belgian pits.

Pit	1877-1892	1908	
1	65 per cent.	17 per cent.	
2	50 „	22 „	Lamps improved.
3	23 „	10 „	
4	41 „	50 „	Lamps not improved.

Sir Josiah Court in 1891 and 1892 examined miners in Derbyshire and Durham, and found among stallmen and holers a percentage affected of 45.75, while among all the miners he examined (2,114), the percentage was only 18.8. Stassen among 11,000 Belgian underground workers found objective signs in 4.2 per cent.; among 4.205 coal-getters he found signs in 33.2 per cent. at the beginning of the shift and in 46.6 per cent. at the end. The figures given by Nieden, Dransart, Coppez, and Llewellyn are quoted. The conclusion reached is that a large proportion of underground miners while still at work exhibit signs of miners' nystagmus which, *if complained of*, might establish a claim for compensation, and, further, that the evidence shows that where lighting has been improved the percentage of miners' nystagmus in safety-lamp pits is less than before.

The report is particularly interesting on the subject of the duration of the incapacity in this country and elsewhere. Thus, in France, Belgium, and Germany, most of the incapacitated men commence work on the surface either at once or at latest within a few weeks, and, with few exceptions, resume their original work within two or three months. Stassen has only seen three cases incapacitated for longer than twelve months. He has ascertained the periods before return to work to be as follows:—

Period off work.	Percentage
0 to 1 month	28
1 month to 3 months	50
3 months to 6 months	20
6 months and over	2
	100

Compare this with the figures for this country (1921 and 1922), as furnished by the Yorkshire Coal Owners' Mutual Indemnity Company.—The total number of certificates sent to them in the two years was in 1921, 221 and in 1922, 558—that is to say, an increase of 150 per cent. At the end of 1920 and 1921 the claims on all the coalfields which had lasted over twelve months and which still remained undetermined were as follows:—

	1920.	1921.
Over 1 year and under 2	952	1,363
,, 2 years ,, ,, 5	932	1,457
,, 5 ,, ,, ,, 10	442	556
,, 10 ,, ...	56	89

After contrasting the relative prevalence of compensated incapacity from nystagmus in Germany, Belgium, and France,

where it is small (0.03 to 0.3) with that in England, where it is great and increasing, the report points to factors as accounting for the increase in this country the widening of the basis for claims (July 9, 1913) by the alteration of the nomenclature of the disease in the Schedule from "Nystagmus" to "The disease known as Miners' Nystagmus . . . whether the symptom of oscillation of the eyeballs be present or not," whereby cases claiming compensation for psychoneurosis were included. The maximum compensation money was in 1919 increased from 20s. to 35s. a week, in order to compensate the lessened value of money. Next followed trade depression with unemployment and a substantial fall in wages; but the compensation remained unaltered. The Committee concludes that while there is no evidence of any increase in the percentage of miners affected with nystagmus, there has been in this country very marked increase in the number of fresh claims for compensation, and in the duration of the incapacity claimed for it, while no corresponding increase has taken place in other countries. The Committee considers that the standard for compensation in vogue in this country lends itself to misuse, and that it should be altered. It is considered that miners should be thought to have nystagmus sufficiently severely to be certified for compensation if oscillation of the eyes is constantly present even when the miner is looking at or below the horizontal level in daylight, or if it persists for five minutes in a good light with the eyes at the horizontal level after it has been induced by darkening the room and using suitable exercises. Machinery should exist for assessing periodically the incapacity present for which compensation is being paid and upon which the amount of compensation should be based. The Committee concludes that most cases of miners' nystagmus are only partially incapacitated, and benefit physically and psychologically by work. Some require work above ground; others are fit for suitable work below ground. A few exceptional cases with short mining experience should never return to work below ground. Such men should in their own interests be drafted out of the industry at once.

2. *Relative importance of errors of refraction in miners' nystagmus.*—Snell, Dransart, Romiée, Stassen, and Coppez attributed no importance to errors of refraction in miners' nystagmus, but Anderson assigned to them the principal cause. Pooley examined the refraction in 130 cases of miners' nystagmus and of 122 miners not so affected. The ultimate analysis of the figures obtained from these sources showed that "errors of refraction in themselves have no effect whatever on the incidence of miners' nystagmus or on the age at which incapacity commences from miners' nystagmus."

S. S.

Senile Cataract: Methods of Operating. By W. A. FISHER, M.D., F.A.C.S. Published by Chicago Eye, Ear, Nose and Throat College, Chicago, Ill. 256 pp. 1923.

In the preparation of this book Fisher has had the assistance of five confrères; indeed only three of the nine chapters of which it is composed are from his pen. His collaborators are Professor E. Fuchs (Vienna), and I. Barraquer (Barcelona), Lt.-Col. Henry Smith (London), Drs. H. T. Holland (Shikarpur, India), and J. W. Wright (Columbus, Ohio). Each of these writers describes the methods of operating which he has practised and which he advocates, and Barraquer, whose contribution to the volume is the most extensive, prefaces his description by a few pages on the "Evolution and Critique of the Cataract Operations." In the final chapter entitled "A method of acquiring technique," Fisher describes various operations for extraction of cataract as performed on the dead eye. For this purpose he prefers the eye of a kitten about six weeks old, because the diameter of its cornea is approximately the same as that of the human cornea (11 mm.).

The book is copiously illustrated, printed in excellent type and has a full index. As a guide to the technique employed by the writers it should prove helpful. We imagine, however, that the young surgeon who consults it will experience considerable difficulty in determining which method to adopt.

CORRESPONDENCE

WERNICKE'S PUPILLARY REACTION

To the Editor of THE BRITISH JOURNAL OF OPHTHALMOLOGY

SIR,—In the last number of the Journal I observe a note on "Wernicke's Pupillary Reaction," by Dr. J. D. Cummins. If the author of this note will refer to the Presidential address which I had the honour to deliver before the Ophthalmological Society of the United Kingdom (*Trans. Ophthal. Soc.* U.K., Vol. XLI, 1921) he will see that I there indicated a method of testing this reaction of the pupil which is identical with that which he now suggests, with the exception that I employed the two eyes successively in making the observation, and not the two halves of the same eye. My method is, I consider, superior to that of Dr. Cummins in that the anatomically corresponding half of each retina successively receives an identical stimulus, the eye in each observation being that nearest to the source of lateral daylight illumination; such an equality of stimulus cannot be obtained when it is received first on the nasal half and secondly on the temporal half of the retina of the same eye, which during Dr. Cummin's first observation is next to the window, and during the second remote from it and to some extent shaded by the features of the nose and face.

Yours truly,

LONDON, *Sept.* 6, 1923. J. HERBERT FISHER.

NOTES

Deaths

IT is with deep regret that we note the death of GUSTAVUS HARTRIDGE, aged 73 years, on September 8, at his residence, Wimpole Street, London, his wife predeceasing him by three weeks. He was the son of James Hartridge, Yalding, Kent, and married the eldest daughter of Colonel Murray, 42nd Highlanders (Black Watch). Hartridge received his medical education at King's College, London, and was Hunterian Prosector at the Royal College of Surgeons of England. He was Assistant Surgeon to the Central London Ophthalmic Hospital. Indeed, he filled several hospital appointments. He was Ophthalmic Surgeon to and Lecturer on Ophthalmology at the Westminster Hospital, Surgeon to the Royal Westminster Ophthalmic Hospital, and finally, Ophthalmic Surgeon to St. Bartholomew's Hospital, Chatham. He wrote several books, of which the best known is the phenomenally successful 'Manual for Students on the Refraction of the Eye,' which has reached its sixteenth edition. Hartridge had been Vice-President, Secretary, and a member of the Council of the Ophthalmological Society of the United Kingdom.

We regret to announce the death towards the end of August of ALLAN E. TAYLOR, once resident ophthalmic officer at Leeds Infirmary, from heart failure after heat-stroke at sea. He took a temporary commission in the R.A.M.C. in 1914, was promoted captain, and served as officer in charge of Ophthalmic Centre No. 82.

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Appointments

Dr. S. M. MORRIS, formerly professor of ophthalmology, otology, and laryngology, has been appointed professor of ophthalmology in the University of Texas (Department of Medicine), Galveston, U.S.A.

PROFESSOR HALLAUER has been appointed to the chair of ophthalmology in Basle, vacated by the departure of Professor Vogt for Zurich.

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Prof. Uhthoff

PROFESSOR UHTHOFF, who recently resigned the chair of ophthalmology at Breslau, celebrated his seventieth birthday on July 31.

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Trachoma Compaign

DR. CHARLES KOBERT, director of the trachoma bureau of the State Board of Health, has left Louisville to tour south central Kentucky treating the victims of trachoma. Clinics will be held in Butler,

Ohio, Edmonson, and Grayson. The mule-drawn wagon, by which Dr. Kobert travels, will return to Louisville late this autumn.

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Insurance Acts: Ophthalmic Benefit

At a meeting of the Council held during the recent meeting of the British Medical Association at Portsmouth, DR. WALLACE HENRY, chairman of the representative body, gave a report of the conference of ophthalmologists called to consider the provision of ophthalmic benefit under the National Insurance Acts. The conference agreed that it was undesirable that patients who required ophthalmic benefit should be sent for treatment by opticians, and approved of a scheme of insured persons, and for the drawing up of a list of ophthalmic surgeons willing to give treatment in accordance with this arrangement. In order to work out the details a committee was appointed of Dr. Brackenbury and Dr. Wallace Henry, as representating the Association generally, and of Mr. T. Harrison Butler, Mr. R. J. Coulter, Mr. N. Bishop Harman, Colonel Ransom Pickard, Mr. G. H. Pooley, and Mr. W. G. Sym, as representing ophthalmologists.

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Bulletin de la Ligue Contre le Trachome. No. 1, 1923

DURING the war troops were brought by France from her Asiatic and African colonies and protectorates. Large numbers of them were trachomatous. Curative and prophylactic measures were instituted and special regulations enacted to cope with the disease. Shortly after the armistice special efforts were made in the countries in which trachoma is prevalent to institute prophylactic and other treatment on a large scale.

At the annual congress of the Société française d'Ophtalmologie, 1922, a proposal to form an anti-trachoma league was brought forward by Drs. C. Nicolle and Cuénod, and was well received. In September of the same year this proposal was more fully considered at a Colonial Congress of Public Health, at Marseilles, and at a meeting held on April 30, 1923, at the Pasteur Institute in Paris, the league was definitely established.

The first number of the *League Bulletin* contains an account of the proceedings which led to the formation of the league; also the rules and regulations which have been adopted. The first list of officers is:—

President; Dr. Charles Nicolle: Vice-Presidents; Drs. Morax and Aubaret: Treasurer; M. Tendron: Secretary; Dr. Paul Petit. The headquarters are at the Pasteur Institute, and the secretarial office is at 19, Rue de Bourgogne, Paris.

The Committee will be grateful for information from ophthalmologists concerning their work among the subjects of trachoma and ask especially for particulars as to the origin and mode of contagion, the nationality of the individuals, and the conjunctival conditions obtaining in those in immediate contact with them.

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Abbreviated Journal Titles

WE think it is worth while to print the abbreviations adopted in this journal for some of the titles of the journals to which reference is frequently made. The editors would be very grateful if contributors would adopt them. They correspond closely with those recommended in Mellish (*The Writing of Medical Papers*, W. B. Saunders Co., 1922), a book well worth its cost to any writer of medical papers:

Acta Ophthalmologica—Acta Ophthal.; American Journal of Ophthalmology—Amer. Jl. of Ophthal.; Annali di Ottalmologia—Annal. di Ottal.; Annales d'Oculistique—Annal. d'Ocul.; Archivio di Ottalmologia—Arch. di Ottal.; Archiv für Anatomie und Physiologie—Arch. f. Anat. u. Physiol.; Archiv für Augenheilkunde—Arch. f. Augenheilk.; Archives d'Ophtalmologie—Arch. d'Ophtal.; Archives für Ophthalmologie—Arch. f. Ophthalm.; Archives of Ophthalmology—Arch. of Ophthal.; Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie—Beitr. z. path. Anat. u. z. allg. Path.; Berliner klinische Wochenschrift—Berl. klin. Wochenschr.; British Journal of Ophthalmology—Brit. Jl. of Ophthal.; British Medical Journal—Brit. Med. Jl.; Centralblatt or Zentralblatt—Centralbl.; Deutsche medizinsche Wochenschrift—Deutsch. med. Wochenschr.; Journal of Anatomy and Physiology—Jl. of Anat. and Physiol.; Klinische Monatsblätter für Augenheilkunde—Klin. Monatsbl. f. Augenheilk.; Ophthalmic Review—Ophthal. Rev.; La Clinique Ophtalmologique—La Clin. Ophtal.; Proceedings of the Royal Society of Medicine—Proc. of the Roy. Soc. of Med.; Revue Générale d'Ophtalmologie—Rev. Gén. d'Opht.; Royal London Ophthalmic Hospital Reports—Roy. Lond. Ophthal. Hosp. Reps.; Transactions of the American Ophthalmological Society—Trans. Am. Ophthal. Soc.; Transactions of the Ophthalmological Society of the United Kingdom—Trans. Ophthal. Soc. U.K.; Wiener medizinsche Wochenschrift—Wien. med. Wochenschr.; Zeitschrift für Augenheilkunde—Zeitschr. f. Augenheilk.

This short list does not pretend to be in any way complete but should serve as a guide to the method of abbreviating adopted in this journal.